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BRAIN:

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FOR THE NEUROLOGICAL SOCIETY OF LONDON

BY

A. DE WATTEVILLE

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AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1899,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1898.

LONDON:
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Neurological Society of London.

REPORT OF COUNCIL.

FEBRUARY 2, 1899.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The members now number 206.

The Inaugural Address was delivered at the Annual General Meeting on January 13, 1898, by Mr. Victor Horsley, F.R.S., on "The Degree of Discharge of Different Nerve Centres."

The vacancy caused on the list of Honorary Members by the death of Professor Du Bois Reymond, of Berlin, has been filled by the election of Professor Hitzig, of Halle, formerly one of the Corresponding Members of the Society, and the vacancy thus caused on the list of Corresponding Members has been filled by the election of Professor Gustaf Retzius, of Stockholm.

A feature in the year's programme has been a visit, in April, to Cambridge, where, at the invitation of the Vice-Chancellor of the University, Dr. Alex. Hill, formerly President of the Society, a Physiological Meeting was held. At the conclusion of the meeting the Society was most hospitably entertained by the Vice-Chancellor. This was the first occasion on which the Society has met out of London.

As during last year, the Proceedings have been published at greater length in "Brain" than was the custom in former years, and an abstract appended to this Report gives some idea of the value and character of the work of the Society during the past year.

The Council reports with regret the resignation of the Senior Secretary, Dr. Frederick Mott, after four years of valuable service rendered to the Society.

Proceedings of the Society during the year 1898.

January 13th, Mr. VICTOR HORSLEY, F.R.S., Inaugural address, "The Degree of Discharge of Different Nerve Centres," *Brain*, vol. xxi., part lxxxiv.

February 24th, Dr. DONKIN (1) "Spastic Paraplegia in a Child aged 4 years;" (2) "Functional Hemiplegia with Hemianæsthesia in a Man aged about 50 years;" (3) "A Case of Paralysis Agitans." Dr. MOTT, "A Case resembling Scleroderma in which there was Diminished Sensibility over the Area supplied by the Two Inferior Divisions of the Fifth Cranial Nerve, with loss of taste over the anterior two-thirds of the tongue, and loss of smell." Dr. HERBERT HAWKINS, "Case of Peroneal Type of Muscular Atrophy." Dr. BEEVOR, "Case of Syphilitic Spinal Meningitis." Dr. LEONARD GUTHRIE, "Case of Unilateral Syringo-myelia with Bulbar Symptoms." Dr. STEPHEN MACKENZIE, "A Case of Nodding Spasms." Dr. ORMEROD, "A Case of General Athetosis." Dr. F. E. BATTEN, "Friedreich's Disease with Mental Defect." Dr. ALDREN TURNER, "A Case of Diplegia Facialis, with paralysis of both external recti, defect in finer movements of hands, spastic gait and double club-foot."

April 23rd, Dr. ALEX. HILL, "Some Specimens of Granules." Professor MACALISTER, "The Race-Characters of Australian Brains." Dr. LANGLEY, "Some Points relating to the Sympathetic System." Dr. GASKELL for Miss ALCOCK, "The Distribution of the Facial Nerve in Ammocœtes." Dr. F. E. BATTEN (1) "Nerve Terminations in Muscle Spindles;" (2) for Dr. SIHLER, of Ohio, "Motor Nerve Termination within the Muscle Spindle." Dr. MOTT, "Degeneration of the Neuron."

June 30th, Dr. C. E. BEEVOR opened a discussion on "The Accurate Localisation of Intra-cranial Tumours," and Drs. BYROM BRAMWELL, MICHELL CLARKE, and SHARKEY took part in the discussion.

October 27th, Drs. ORMEROD and F. E. BATTEN, "Idiopathic Muscular Atrophy (Landouzy-Déjérine type) in Mother and Son." Dr. BEEVOR, "Unilateral Wasting and Paralysis of the Lower Half of the Trapezius." Dr. SINIGAR, "Case of Idiopathic Muscular Atrophy." Dr. F. W. MOTT, "Arsenical Neuritis from Local Action of the Poison followed by general Degenerative Effects upon the Nervous System." Dr. ORMEROD, "A Case of Hereditary Chorea." Dr. LUNN and Mr. BALLANCE, "Case of Removal of Cerebellar Tumour, three years and eleven months after the operation" (case shown at a meeting of the Society eighteen months ago). Mr. LAKE and Mr. BALLANCE, "Case of Recovery from Temporo-Sphenoidal Abscess." Dr. BEEVOR and Mr. BALLANCE, "Case of Removal of Subcortical Cerebral Tumour." Dr. JAMES TAYLOR and Mr. BALLANCE, "Case of Trigeminal Neuralgia, in which removal of the Gasserian Ganglion was followed by complete relief."

December 15th, Dr. F. W. MOTT, "Changes in the Brain, Muscles, and other Organs, found in persons having died with prolonged epileptiform convulsions." Dr. F. E. BATTEN, "Congenital Abnormalities in the Nervous Systems of Three Children." Drs. H. H. TOOTH and RISIEN RUSSELL, "Acute Myelitis with Bacteriological Examination."

BALANCE SHEET, 1898.

Cr. NEUROLOGICAL SOCIETY. **Dr.**

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Subscriptions for current year	192	4	0
Do. in arrears	12	0	0
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				£310	12	7

Audited and found correct, December 6, 1898,

CHARLES E. BEEVOR, *Hon. Treasurer.*

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RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

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*** Members are requested to communicate with the Secretaries when corrections are necessary.

—:o:—

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.
Pres., President.
V.P., Vice-President.
Tr., Treasurer.
Sec., Secretary.
C., Member of Council.
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1892 Bristowe, Hubert Carpenter, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.
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1888 Brown, Sanger, M.D., 100, State Street, Chicago.

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,, Bruce, J. Mitchell, M.D., F.R.C.P., 23, Harley Street, W.

1898 Bruce, Samuel Noble, M.R.C.S.Eng., 43, Kensington Gardens Square.

O.M. Brunton, T. Lauder, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-8.)

1895 Bryan, Frederick, M.B.Dunelm, M.R.C.P., The Asylum, New Southgate, N.

,, Bryant, John Henry, M.D.Lond., L.R.C.P., Guy's Hospital, S.E.

1890 Bury, Judson Sykes, M.D., F.R.C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-89; C., 1886-7, 1891-5.)

1898 Caley, Albert, M.D.Lond., M.R.C.P., 24, Upper Berkeley Street, W.

1898 Campbell, Harry, M.D., B.S., F.R.C.P.Lond., 23, Wimpole St., W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

1898 Clinch, Thomas Aldous, M.D.Edin., Durham County Asylum, Winterton, Ferryhill, Co. Durham.

O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 Colman, W. S., M.D., F.R.C.P., 22, Wimpole Street, W. (Sec., 1899.)

,, Corner, Harry, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1898 Crookshank, F. Graham, M.D.Lond., M.R.C.S., L.R.C.P., County Asylum, Berry Wood, Northampton.

1894 Cuming, James, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.

1895 Davidson, Andrew, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 Dawson, William R., M.D., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

1894 Dean, H. Percy, M.D., F.R.C.S., 69, Harley Street, W.

1898 Dodd, Henry Work, F.R.C.S.Eng., 136, Harley Street, W.

O.M. Dreschfield, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P., 1898-9.)

,, Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Symons, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894: V.P., 1892-3; C., 1886-90-95.)

Elected

O.M. Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

,, Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.P., 1897-8; C., 1896.)

,, Gee, Samuel, J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1898 Goodliffe, John Henry, M.D.Aberd., County Asylum, Berry Wood, Northampton.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, Dundas, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. Gunn, R., Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899.)

1895 Guthrie, Leonard, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., The Oaks, Longlands Road, Sidcup.

1897 Harris, Wilfred J., M.D., M.R.C.P., 7, Craven Mansions, Craven Terrace, W.

1898 Hartley, W. Darley, M.D. (Dunelm), M.R.C.P. (Lond.), "Mayfield," Gilmore Hill Road, Cape Town, South Africa.

,, Haslett, John Handfield, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 Head, Henry, M.D., M.R.C.P., 61, Wimpole Street, W.

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896-7; C., 1892-3, 1899.)

1889 Huggard, William R., M.D., F.R.C.P., Davos Platz, Switzerland.

1896 Hulme, George Frederick, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 Humphry, Laurence, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.D., M.R.C.P., 6, Manchester Square, W.

Elected

1892 Jones, Henry Lewis, M.D., F.R.C.P., 61, Wimpole Street, W.

1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W.

1896 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 Langdon-Down, Reginald L., M.B., B.C.Cantab., M.R.C.P.Lond., 81, Harley Street, W.

O.M. Langley, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge (C., 1898-9.)

1888 Lawford, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

1897 Lewis, H. Wolseley, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

,, Lowenthal, Max, M.D., M.R.C.P., 84, Princes' Road, Liverpool.

1898 MacCormac, John M'Gee, M.D.Durh., L.R.C.P., L.R.C.S.Edin., 31, Victoria Place, Belfast.

,, McAldowie, Alexander M., M.D.Aberd., 6, Brook Street, Stoke-on-Trent.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896-8.)

1896 Mackintosh, Ashley W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, Mann, J. Dixon, M.D., F.R.C.P., 16, St. John's Street, Manchester.

1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 May, W. Page, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. Mercier, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C. 1899.)

1897 Meyer, Adolf, M.D., Clark University, Worcester, Mass., U.S.A.

O.M. Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895-97.)

1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.

1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-8; C., 1899.)

1894 Murray, George R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.

Elect
1889 Murray, H. Montague, M.D., F.R.C.P., 27, Saville Row, W.

O.M. Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.
,, Nicolson, David, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.
1888 Niermeyer, J. H. H., M.D., Amsterdam.
,, Norman, Conolly, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.
,, Ogilvie, Leslie, M.B., M.R.C.P., 46, Welbeck Street, W.
,, Oliver, Thomas, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.
O.M. Orange, Wm., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.
,, Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.
,, Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-7.)

,, Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)
1895 Parkinson, John Porter, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.
1896 Paterson, Donald Rose, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.
1895 Pinder, George, M.B., B.C., Seafield House, Ramsey, Isle of Man.
1888 Pitt, G. N., M.D., F.R.C.P., 15, Portland Place, W.
O.M. Poore, G. Vivian, M.D., F.R.C.P., 32, Wimpole Street, W. (C., 1898-9.)
O.M. Purves, W. Laidlaw, M.D., 20, Stratford Place, W.

1894 Ransom, William B., M.D., F.R.C.P., The Pavement, Nottingham.
1890 Rayner, Henry, M.D., M.R.C.P.Ed., 2, Harley Street, W.
O.M. Reid, E. W., M.B., University College, Dundee.
1897 Reissmann, Charles H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.
1893 Rennie, George E., M.D.
1895 Reynolds, Ernest S., M.D., F.R.C.P., 23, St. John Street, Manchester.
1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.
1891 Rivers, W. H. R., M.A., M.D., M.R.C.P., St. John's College, Cambridge. (C. 1897-8.)
1898 Robertson, William Ford, M.D., C.M.Edin., 12, Bristo Place, Edinburgh.
1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.
,, Ruffer, Marc Armand, M.D., Cairo.
1889 Russell, J. S. Risien, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-9.)

O.M. Sanderson, J. Burdon, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.
,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square, (Pres., 1897; V.P., 1895; C., 1886-90, 1898.)

Elected	
1888	Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.
O.M.	Schäfer, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)
,,	Semon, Sir Felix, M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899.)
1898	Seward, Wm. Joseph, M.B.Lond., Colney Hatch Asylum, N.
O.M.	Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)
,,	Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C. 1892-95.)
1894	Shuttleworth, George E., M.D., Ancaster House, Richmond.
1898	Simpson, Francis O., L.R.C.P.Lond., M.R.C.S.Eng., Hawkhead Asylum, Paisley, Scotland.
1895	Smith, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.
1887	Smith, R. Percy, M.D., F.R.C.P., 36, Queen Anne Street, W.
1895	Smith, Telford, M.D., B.S., Royal Albert Asylum, Lancaster.
,,	Spicer, William T. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.
,,	Stansfield, Thos. E. K., M.B., Claybury Asylum, Woodford, Essex.
,,	Stanley, Douglas, M.D.Edin., 9, Easy Row, Birmingham.
1892	Starling, Ernest Henry, M.D., F.R.C.P., B.S., 8, Park Square, N.W.
1897	Stewart, Purves, M.A., M.D., M.R.C.P., 133, Harley Street, W.
O.M.	Stewart, Sir T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.
,,	Stirling, Wm., M.D., D.Sc., Owens College, Manchester.
1897	Stoddart, Wm. H., M.D., B.S., M.R.C.P.Lond., Bethlem Royal Hospital, Lambeth, S.E.
1895	Stout, G. F., M.A., St. John's College, Cambridge.
1887	Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.
O.M.	Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)
1889	Sutherland, Henry, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.
1888	Syers, Henry W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.
1898	Taylor, F. R. P., M.D., B.S.Lond., Darenth Asylums and Schools, Dartford, Kent.
1891	Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W.
1898	Thomson, H. Campbell, M.D., M.R.C.P., 34, Queen Anne Street, W.
1889	Thorburn, Wm., F.R.C.S., The Limes, Nelson Street, Manchester.
1892	Titchener, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.
O.M.	Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-9.)
1892	Trevelyan, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.
1892	Tuckey, Charles Lloyd, M.D., C.M., 88, Park Street, Grosvenor Square, W.
1892	Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

Elected

1891 Tuke, Thomas Seymour, M.B., M.R.C.S., Chiswick House, Chiswick.

,, Turner, William Aldren, M.D., F.R.C.P., 13, Queen Anne Street, W.

1898 Turney, Horace G., M.B., M.Ch.Oxon, F.R.C.P.Lond., 68, Portland Place, W.

O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.

1888 Voorthus, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

1895 Wade, Arthur L., M.D., Somerset and Bath Asylum, Wells.

1894 Walker, A. Stodart, M.B., 30, Walker Street, Edinburgh.

O.M. Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (V.P., 1899; C., 1894-7.)

O.M. Ward, J., D.Sc., Trinity College, Cambridge.

,, Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 Washbourn, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. Watteville, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-9; Sec., 1886-9; Editor of *Brain.*)

1897 Weber, F. Parkes, M.D., F.R.C.P., 19, Harley Street, W.

,, West, Samuel, M.D.. F.R.C.P., 15, Wimpole Street, W.

,, White, Ernest W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. White, W. Hale, M.D., F.R.C.P., 65, Harley Street, W. (C., 1897-9.)

1894 Whiting, Arthur J., M.D., M.R.C.P., 142, Harley Street, W.

1889 Wiglesworth, Joseph, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1894 Williamson, Richard T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

1896 Wilson, Albert, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, Wood, Guy M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 Wood, T. Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

1898 Young, Robert Arthur, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.

BRAIN.

PART I., 1898.

Original Articles and Clinical Cases.

HÆMORRHAGE INTO PONS, SECONDARY LESIONS OF LEMNISCUS, POSTERIOR LONGITUDINAL FASCICULI, AND FLOCCULUS CEREBELLI.

BY SAMUEL GEE, M.D., AND H. H. TOOTH, M.D.

The case here reported presents, we think, noteworthy anatomical features in connection with the destination of the lemniscus or fillet, the relations of the posterior longitudinal bundles with the cranial nerve nuclei, and also the probable destination of the two sets of fibres, afferent and efferent, constituting these bundles.

Points of clinical interest also attract attention, such as a hæmorrhage due to diseased arteries in a young woman aged 21, with advanced interstitial changes in the kidneys, and hypertrophy of the left ventricle.

Sarah P., aged 21 years, unmarried. Nothing can be discovered in her family or previous history throwing any light upon the cause of her illness. There was a doubtful notch in one of the upper central incisors.

September 14, 1896.—She was in good health until this day. At 3.50 p.m., while at work, she suddenly called out, and was seen by her companions to be "in a fit." She became very blue, and probably lost consciousness; but no more facts can be obtained. At 7.30 p.m. she was seen at home by some friends who state that she could not speak. At 9 p.m. she spoke, saying the name of one of her bystanding friends. Asked if she were in pain, she put her hand to her head. The right hand "was constantly twitching;" left hand and arm lay motionless. Vomited about six times through the night. Passed urine naturally. Complained of feeling hot. Slept with both eyes closed; breathing very heavy.

September 15.—Admitted into hospital. Stout, healthy-looking girl. Lies on right side. Temperature normal throughout illness, except on September 16, and September 22, when it rose to 100°.

Understands what is said to her, but what she says is for the most part unintelligible; the defect in speech was deemed to be an imperfect articulation, not aphasia.

Paralyses:—Eyes: Both pupils small, right smaller than left; right reacts to light and accommodation sluggishly; left, promptly. Movements upwards and downwards are good. Some divergent strabismus of right eye on looking upwards. Lateral movements completely paralysed in both eyes; she cannot look to right or to left; nor can she converge at all. Some nystagmus in left eye.

Masticating muscles: See notes next day.

Face: Completely paralysed on right side; cannot close right eye at all.

Soft palate apparently not at all affected.

Tongue protruded pointing to the right.

Arms: Right fairly strong. Left almost powerless; she can just move it, but the movements are uncertain and erratic; no reflexes obtained.

Legs: Left leg rather weak, no more. Both patellar reflexes present.

Sensation: Impaired over whole left side of body.

No physical signs of disease in chest or abdomen. Pulse tension raised; artery distinctly thickened.

At 7.45 p.m. she was found snoring and could not be roused. This had passed off by 8 p.m.

September 16.—Paralyses: Power impaired in right masseter, temporal and internal pterygoid muscles. Articulation still affected.

Sensation: Impaired on both sides of the face, most so on upper part of left side. Both conjunctivæ and sclerotics are anæsthetic. [This probably means, anæsthetic on both sides of the body.] Right cornea is anæsthetic, and there are conjunctivitis and keratitis. Sensation impaired on left side of neck and trunk, and on left arm and leg. No loss of sensation in tongue or palate.

Ophthalmoscopic examination showed nothing unnatural, unless it were slight swelling of the disc, and some blurring of vessels at edge of disc.

Pulse 132, full volume, tension raised, artery thickened. Complains of headache.

Urine 1010 sp. gr., thick cloud of albumen, no sugar. By microscope, triple phosphates, and bladder epithelium. Some tendency to retention of urine, rendering use of catheter necessary now and then.

September 17.—Patient says she remembers her attack; was unconscious a little while. Before the attack she had pins and needles in both hands. When she came to her senses she had no headache.

Paralysis:—Left arm and leg; incomplete in both, but much greater in arm than in leg. Complete in muscles supplied by facial and trigeminal nerves on right side. Tongue deviates to right, but less so than on admission. Articulation defective. Complete in external and internal recti of both eyes. A doubtful weakness of left levator palpebræ. No paralysis of palate. Some tendency to vertical nystagmus in left eye. Swallows well. Respiratory movements natural.

Electrical re-actions normal, except that the right facial muscles do not react to faradic current.

Anæsthesia:—Of the whole left side, excepting a small patch on dorsum pedis. In face, anæsthesia most marked in left upper half of face and scalp. Some impairment of sensibility on right side of face. Complete anæsthesia of right cornea; partial of left. Sensation of tongue, palate and inside of mouth natural. She thinks she hears better with right ear than left, but this is doubtful.

Retention of urine. Urine clouded by a few pus globules.

Temporary swelling of submaxillary salivary glands about this time. Mind pretty clear. Pulse very frequent, 132.

September 18.—Passing urine naturally; much pus; catheter has been used.

September 22.—Tongue protruded straight; both sides react to faradism. An apparent ptosis of left eyelid, which disappears on looking upwards, and is probably due to the ophthalmia.

September 23.—A large patch of superficial keratitis of right cornea and discolouration of iris. Right eyelid swollen and red; much conjunctivitis. Retention of urine has recurred.

September 25.—Mental condition not so good; she cries for her mother in a childish way. Fundus of both eyes normal by ophthalmoscope.

September 28.—Has been gradually getting more and more drowsy the last few days. Lies with mouth open; breathes heavily. Pulse hard, 130. Respirations 24. Temperature subnormal. Eyes as before. Right facial muscles do not respond to the strongest faradic current. Faradic reactions in limbs normal. Some wasting of left arm and leg. Excessive patellar reflex, and ankle clonus on both sides. Sensation not so completely lost on left side as was the case a week ago. Taste seems to be absent on the anterior part of the tongue, normal on posterior part. Says she does not smell so well as formerly. Sight unaffected. Hearing unaffected.

September 30.—Much more drowsy, even falling asleep while being faradised. After this date her pulse and breathing gradually failed; she became unconscious and died on October 3.

The temperature chart shows a rise to 100° F. on the day after admission, and again to 100° on September 22. Between these dates, and after the 22nd, the temperature was normal.

POST-MORTEM EXAMINATION.

October 4, 1896.—Very thin girl, very pale, teeth good, rigor mortis in legs, not in arms.

Head.—Cranial bones natural. Dr. Gee examined head himself, and dictated following note:

"Longitudinal sinus empty. Convolutions flattened, but no sign of tubercle nor of exudation. No tumour on convexity. The anterior tip of temporo-sphenoidal lobe is adherent. The pons looks a little baggy. On the right middle cerebral artery at its first bifurcation about one inch from its commencement, is an opaque yellow patch surrounding the artery (probably atheroma). No embolus. There is more than the ordinary amount of water in ventricle. Septum lucidum natural. The lyra seems decidedly soft; ? ante-mortem. Plexuses quite natural. The corpora striata and optic thalami natural. Cerebellum externally natural. On opening the valve of Vieussens close behind corpora quadrigemina, and on right side is a reddish-brown protrusion about the size of a cherry stone. The median groove on floor of fourth ventricle is pushed to the left by the tumour. The mass spoken of is above the auditory striæ. All the nerves at base of brain look natural and are not involved in any effusion. Sylvian aqueduct natural."

Lungs.—Both together 20 oz. Old adhesions at apices. No tubercle.

Heart.—11 oz. Pericardium contained 5 oz. brown turbid fluid. Vessels of pericardium injected. Slight exudation of lymph at upper part of auricles. Tricuspid valve natural, also pulmonary. Mitral valve admitted tips of two fingers, looked thickened, a few vegetations on auricular surface. Aortic valves competent to water test; certainly roughened on ventricular surface, and free edges of lunules eroded in places. Heart muscle pale and rather soft. No fatty striation. Left ventricle somewhat hypertrophied.

Kidneys.—2 oz. each. Very small and granular. Capsule adherent and thickened. Cortex scarcely to be seen. Much fat in hilum. Ureters natural, not dilated. Bladder showed no sign of inflammation, except at urethra, which was probably caused by use of catheter. Some pus in vagina and some injection of its mucous membrane.

Summary.—Granular kidneys. Hypertrophy of left ventricle. Endocarditis, ? acute ulcerative. Acute pericarditis.

GENERAL DESCRIPTION OF THE HÆMORRHAGE.

This is roughly fusiform, with its longest and narrowest end brainwards. It is situated on the right side, but encroaches a little to the left, having destroyed a considerable length of the median raphe, at the same time pushing aside and dislocating structures immediately to the left of the raphe. The greatest diameters lie at about the junction of the middle with the lower third of the pons (see figs. 1 and 2), and dorsally it bulges into the fourth ventricle, pushing up its floor about a quarter of an inch above the general level, without, however, having burst into the ventricle. Ventrally it erodes in a very irregular manner nearly down to the pyramidal fibres, some of the most dorsal of which may be involved (figs. 11 and 12). Its spinal or posterior end follows the line of the sixth nucleus, so that except for a very small posterior portion, the whole of the right sixth nucleus may be said to be destroyed. Brainwards the line of hæmorrhage seems to follow and be limited to the right lemniscus or fillet, which nearest the main mass of hæmorrhage is almost totally destroyed. It follows the bundle of fibres, as along a line of least resistance, to a point at about the level of the spinal end of the fourth nucleus.

Method of Examination.

The medulla and pons were hardened for about fourteen days in Müller's fluid. Then slices about $\frac{1}{8}$ in. thick were placed in Marchi's fluid for two to three weeks. These after prolonged washing (sometimes over a week) were imbedded in celloidin, and over 200 sections mounted for examination. Of these about one in three were stained by the Pal method in addition. These were valuable for general anatomical purposes, but for the degeneration the Marchi method alone is far superior.

Detailed Examination of the State of the Surrounding Tissues, Nuclei and Fibres Involved.

Fig. 12 is an outline diagram of a section made a little posterior to the greatest diameter of the hæmorrhage. The hæmorrhage here presents a sinuous outline, with an irregular cavity and a number of smaller fissures due probably to shrinkage under the hardening reagents. The edge is sharply marked by what is evidently recent hæmorrhage for a thickness of from $\frac{1}{16}$ to $\frac{1}{8}$ in. The central mass is of a lighter colour, probably consisting of altered fibrin. It contains a quantity of osmic acid staining fatty *débris*, and also numbers of corpuscles of the size of white corpuscles containing in their substance many black granules of fat. In addition to these are many corpuscular masses, as large as a medium sized ganglion cell loaded with black granules, and presenting in some cases an indistinct clearer space in the centre suggesting a nucleus. They are probably of the nature of phagocytes loaded with degenerated myeline.

Though the arterial walls must have been much damaged no definite aneurysmal dilatations can be seen anywhere. There are, however, several small independent hæmorrhages in neighbourhood of the main lesion on both sides and in the middle line. (Figs. 13 and 14, *h*.)

NUCLEI, FIBRES AND NERVE TRUNKS DIRECTLY DESTROYED BY THE HÆMORRHAGE. (PRIMARY LESIONS.)

Except for a small posterior portion (fig. 16), the whole of the right sixth nucleus is destroyed (figs. 12-15, inclusive). With it, as might be expected, the ascending loop of the seventh is involved, and also the posterior longitudinal bundle. The left sixth nucleus is only slightly if at all affected, and probably only by pressure. The posterior longitudinal bundle on the left side is distinctly degenerated, so presumably some of its fibres must have been injured, but not so completely as on the right side (fig. 12, *lbr*). The hæmorrhage at one point encroaches so much over the middle line as to cut across some of the root fibres of the left sixth nerve (fig. 13). The right lemniscus or fillet is completely involved in destruction. The left cannot be seen to be encroached upon, but it must be so to a certain extent, for there are unmistakable degenerated fibres in it and the lateral fillet above. The transverse fibres of the formatio reticularis (figs. 11, 12, 13, *tr*) are extensively destroyed on the right side, and there is degeneration of them on the left.

The cells of the right motor fifth nucleus are difficult to find, being involved in the hæmorrhage, and such as are found are undergoing fatty changes. The sensory nucleus is also much affected, and in places completely destroyed. The ascending (so called) root of the fifth on the right side, all the way down (figs. 13-21, *Va*), presents secondary degeneration of its ventral third. A lesion, therefore, of the root fibres of the sensory fifth must have existed though such cannot be definitely shown.

SECONDARY DEGENERATION OF CRANIAL NERVES.

Fifth. The *ascending root* on the right side shows the degeneration referred to in the last paragraph (figs. 13-21, *Va*).

The *motor root* fibres seem to be completely degenerated (fig. 11, *Vm*).

Sixth. The root fibres on the right side are absolutely degenerated, as might be expected from the completeness of the primary lesion of the nucleus (figs. 13, 14, 15, *VI*). The most anterior fibres of the left sixth root happening to be involved in hæmorrhage encroaching over the middle line are degenerated (fig. 13). The lesion is very slight, however, only to be shown in a few sections.

Seventh. The position of the hæmorrhage is such that, though the seventh nucleus (figs. 14, 15, *VIIn*) is not directly involved, yet the bundle or loop formed by its fibres, and lying dorso internal to the sixth nucleus, is completely destroyed on the right side. Moreover, the supposed connection between the loop and the third nucleus by way of the posterior longitudinal bundle[1] is also severed. Hence an absolute degeneration of the seventh trunk (figs. 14, 15, *VII*).

The condition of the left seventh is interesting. In figs. 14 and 15, *VIIa*, is shown an obvious but not extensive degeneration of the left seventh trunk. This may be explained by a positive lesion of the most mesial fibres of the ascending loop, but it also appears as if degenerated fibres were passing into the loop at all levels from the posterior longitudinal bundle ventral to it, and from the grey matter of the floor of the ventricle dorsal to it, a structure very rich in medullated nerve fibres (this is further discussed below).

Other Cranial Nerves and Nuclei.

The state of the third, fourth, eleventh, and twelfth nuclei nerves will be considered with the posterior longitudinal bundles.

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[1] Tooth and Turner, "Study of a Case of Bulbar Paralysis, &c." BRAIN, XIV., 1891, p. 493.

SECONDARY DEGENERATION OF THE POSTERIOR LONGITUDINAL BUNDLES.

This is a subject of the greatest interest, for it has long been believed on anatomical and clinical grounds that these bundles of fibres serve as paths of communication between the motor cranial nerve nuclei, and that they may also be the channel by which fibres get from a nucleus of one name into a trunk of another.

This fasciculus is completely destroyed for a short distance on the right side, and extensively so but to a less degree on the left; so that there is a well marked secondary degeneration on both sides. The degeneration is also above and below, and therefore this bundle conducts fibres towards and from the brain and in about equal numbers. (?)

(1) *Degeneration below the Lesion* (*spinewards*).

In figs. 12, 13, 14, and 15 the bundle on the right side is completely destroyed, its situation being marked in fig. 15 by a collection of deeply staining masses of degenerated myeline (*lb*).

In fig. 15 the bundle on the left side presents the familiar triangular shape. The loop of the seventh nerve (*VIIa*), in this and fig. 14, presents a number of degenerated fibres grouped ventrally and dorsally. Their derivation from the posterior longitudinal bundle is suggested above.

In fig. 16, lower down, and now below the level of the hæmorrhage, the posterior longitudinal bundles (*lb*) on either side appear to be taking a ventral course, so that they are now cut almost longitudinally. The lines of degenerated fibres *seem* to pass into the interolivary layer or fillet (see also fig. 17), but that tract of fibres below the lesion may be said to be practically undegenerated.

The sixth nucleus (*VIn*) presents a number of degenerated fibres, many apparently entering the nucleus from the grey matter of the floor of the ventricle dorsal to it.

In fig. 18, at the level of the vagus (*X*) and hypoglossal (*XII*) nuclei, the same mass of degenerated fibres (*lb*) can be seen lying mesially to the twelfth roots and diverging with them in the direction of the olives. The twelfth nuclei show a considerable number of degenerated fibres, which as in the nuclei above may be derived from the posterior longitudinal bundles. As in the case of the sixth nucleus also, fibres seem to be entering dorsally.

At the level of the post-pyramidal decussation of the lemniscus (*dlm*, fig. 19), which is natural and undegenerated, we still find between it and the twelfth roots the degeneration above described, but here it receives the name of the anterior ground bundle (*ag*).

A little lower at the level of the decussation of the pyramids (*D*, fig. 20), at which level also the anterior horns of the upper cord are now visible, the degenerated fibres are leaving the central parts of the section to be disposed round the anterior horns and on the right side still further appearing in the lateral region to be, as in fig. 21, disposed round the lateral horn, or in other words, the nucleus of the spinal accessory.

We are therefore justified in regarding the anterior ground bundle as consisting largely, if not entirely, of the lower descending fibres of the posterior longitudinal bundle, and as such may be the channel of communication between the spinal accessory nucleus and the higher motor cranial nerve nuclei.

(2) *Degeneration above the Lesion.*

On the right side the degeneration is very complete, less so on the left (figs. 10 and 9, *lb*).

Tracing the bundles brainwards the first interesting connection is with the fourth nucleus (fig. 8, *IVn*), which at this level is practically blended with the posterior longitudinal bundle. Its cells are natural, but the nucleus is pervaded with degenerated fibres, no doubt derived from the posterior longitudinal bundle; in fact, there can be said to be very few normal fibres. The issuing trunk (*IV*, fig. 8) naturally consists largely of normal fibres, but among them appear also several degenerated fibres, apparently passing away into the trunk without having entered into connection with cells. We infer from this that, as is the case with the motor nuclei hitherto considered, the fourth nucleus receives many fibres from the posterior longitudinal bundles, and that a few of these fibres do not end in the nucleus, but pass straight on into the trunk. May these serve as a connecting link between movements of the superior oblique and external rectus of the same side?

What has been said of the fourth may be equally said of the third nucleus, which is found to be full of degenerated fibres running in every direction. But more than this, we have here an obvious and complete decussation of fibres (fig. 7, 6 and 5, *III*). Among the roots of the third nerves we find a fair number of degenerated fibres (fig. 6), more on the left than the right side. It is tempting to suppose that these are the crossed fibres originating in the sixth nucleus and innervating the opposite internal rectus, for the purpose of conjugate side movements of the eyes, a connection long supposed to exist on clinical grounds.[1]

The crossing of the fibres from one nucleus to the other can be seen to as far as the third nucleus extends (fig. 5). Above this level there still remains a group of degenerated fibres on the mesial side of each red nucleus (*rn*) (figs. 4 and 3, *lb*). These fibres, however, are now taking a direction which causes them to be cut obliquely as if they were passing in a ventral direction. Moreover, there now appears a small bundle of the same fibres (?) in the region of the pillar of the fornix (figs. 4 and 3, *fd*). The destination of these must be conjectural, possibly it may be the corpus mammillare. The fibres of the so-called red nucleus at this level are taking also an oblique course, apparently passing in a lateral direction dorsal to the corpus subthalamicum (*csm*), and among them are many degenerated fibres, possibly derived from the posterior longitudinal bundle. The ultimate destination of many of the fibres may also be the optic thalamus (fig. 3, *ot*).

So that, to sum up, the posterior longitudinal fibres above the third nucleus seem to send fibres ventrally? to end in the corpus mammillare, laterally with the general trend of the red nucleus fibres, and dorsally to the optic thalamus.

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[1] Hughes Bennett and Savill, "Case of permanent conjugate deviation . . . lesions limited to sixth nucleus, etc." BRAIN, vol. xiv., p. 102.

Secondary Degeneration Posterior to the Lesion (Spinewards) of other Fibres.

(1) In the floor of the fourth ventricle there lie normally numbers of fine medullated fibres, which are specially abundant on the dorsal aspect of the various motor nuclei. These fibres, as above noted, are copiously degenerated below the lesion (see figs. 14-18, *d*). It is of course possible that they may constitute an independent commissural system between the nuclei. But this is scarcely probable, and most likely these fibres belong to the posterior longitudinal bundle system.

(2) There is an unmistakable degeneration of the fibres of the antero lateral region shown in figs. 15-19, *at*. This is the situation of the antero lateral ascending tract of Gowers. It was never supposed that this region contained only ascending fibres, but that it resembled the "mixed zone" of the cord. These efferent fibres would seem to be conducted in the formatio reticularis as indicated in figs. 16 and 17, but their actual origin is not known.

(3) A few fibres of the right pyramid are degenerated (figs. 12, 13, *et seq.*, *p*) which can be traced even into the pyramidal decussation (*D*, fig. 20). It is possible that these may be the remains of a considerable pressure lesion at the line of the original hæmorrhage, for no definite solution of continuity of such fibres can be found in any of the sections. That such a pressure did exist is suggested by the transient hemiplegia noted.

Secondary Degeneration of Cerebellar Fibres.

The white matter of the flocculus on either side is extensively degenerated, and in this respect strongly contrasts with that of the rest of the cerebellum (figs. 13-16, *F*). We have been unable to satisfy ourselves as to the origin of the fibres constituting the "arbor vitæ" of the flocculus, but there is undoubtedly at the level of the maximum lesion an extensive degeneration of the transverse fibres of the formatio reticularis (figs. 11-15, *tr*), and it is obvious that such fibres most probably go to the cerebellum on both sides. They do not, however, appear in the middle peduncle as might be expected, and therefore it is quite likely that they find their way into the flocculus.

Secondary Degeneration of the Lemniscus or Fillet.

The state of this bundle of fibres posterior to the lesion may be disposed of in very few words.

The interolivary layer, the post-pyramidal decussation or decussatio lemnisci, and the funiculi gracilis and cuneatus from which the fillet is believed to arise, are all practically free from degeneration. A few fibres in the interolivary layer are degenerated, but there is reason to believe that they belong to the posterior longitudinal bundles. The fillet, therefore, probably consists of afferent fibres only.

Above the lesion we find that the degeneration of the fillet is complete on the right side, but comparatively slight on the left.

In fig. 11 the main or mesial fillet is still quite replaced by hæmorrhage on the right side. On the left side there is a scattered degeneration in the mesial fillet (*lm*), probably secondary to the encroachment of the hæmorrhage over the middle line shown in fig. 12. On both sides can be seen a marked bundle of degenerated fibres (*ll*) apparently proceeding towards and external to the brachium conjunctivum (*bc*). This is the lateral lemniscus, which anatomically may be regarded as a branch of the mesial, but the fibres of which apparently terminate in the corpus quadrigeminum.

In fig. 10 the hæmorrhage is shrinking to the limits of the right mesial lemniscus. The lateral lemniscus is a marked object in the section; a few degenerated fibres apparently belonging to it seem to cross to the opposite side by way of the lingula (*lg*).

At the level of the posterior corpus quadrigeminum (fig. 9, *cq*) the fibres of the lateral lemniscus appear to enter and lose themselves in the corpus quadrigeminum. The mesial lemniscus is totally degenerated on the right side, and still contains a small mass of hæmorrhage, much less so on the left. Above the lesion (fig. 7), at the junction of pons with crus cerebri, the same description applies.

In fig. 6 at the level of the third nerve roots the mesial lemniscus alters in the shape of its section, and the fibres apparently become more scattered and so occupy a larger area. Moreover, the disposition of the fibres is such that there is tendency to half surround the red nucleus (*rn*). On the left side, which owing to lateral obliquity of section is more anterior than the right, the fibres of the mesial lemniscus are beginning to take on a lateral direction between the corpus geniculatum internum (*cg*) and the substantia nigra (*sn*).

This lateral direction of the fibres of the mesial lemniscus is still more marked in the section above (fig. 5). A few fibres seem to go to the corpus geniculatum, and the remains of the lateral lemniscus can be seen entering the corpus quadrigeminum.

At the level of the posterior part of the optic thalamus (*ot*, fig. 4) the lemniscus fibres are cut longitudinally, indicating a general change in direction, and suggesting that the greater number of them stream away to the side, that is, away from the optic thalamus, between it and the corpus subthalamicum (*csm*), which here replaces the substantia nigra. This point is also well shown in fig. 3, in which the optic thalamus is well represented on the left side. Unfortunately there was not enough of the brain saved to carry these observations higher or more laterally. It is, therefore, possible that the fibres of the lemniscus may all eventually find their way into the optic thalamus. There can be little doubt that a large number end there, but it is highly probable that many are taking the direction indicated in these diagrams to pursue an uninterrupted course to the cortex.

REMARKS.

Reconciliation of Symptoms with Pathological Findings.

The left hemiplegia was no doubt due to pressure on the pyramidal fibres, for the actual damage to the tract is very slight. The hemianæsthesia on the left side might be expected from the very extensive lesion of the right fillet and surrounding structures, formatio reticularis, or tegment (figs. 11, 12 and 13). Mott[1] finds that complete hemianæsthesia does not follow in monkeys after fairly complete degeneration of the lemniscus secondary to lesions of the nuclei funiculi gracilis and cuneatus, but he notes "a distinct lowering of sensibility on the side of the lesion," and that the animal did not use the hand of that side with the same facility as on the other side. So that it would appear that the lemniscus is not the only path of sensory impressions to the brain. This point is confirmed by the experiments of Ferrier and Turner,[2] who regard the tegment as possibly the path of tactile and pain sensations.

The anæsthesia of the upper part of the right face with keratitis follows from the extensive destruction of the right fifth sensory nucleus. The loss of taste on the anterior part of the tongue suggests involvement of the sensory cells of the chorda tympani.

The complete paralysis of the masticatory muscles on the right side might be expected from destruction of the motor nucleus of the fifth, and the consequent secondary degeneration of its root fibres.

The connection between the symptoms and the condition of the sixth nuclei presents certain difficulties. The practically complete destruction of the right sixth nucleus perfectly explains the loss of power of conjugate deviation to the right. But the patient is noted to have had equally complete conjugate paralysis of movement to the left, while the left sixth nucleus is uninjured. The posterior longitudinal bundle on the left side is considerably degenerated, but not so much so as on the right, therefore we may assume that the communication between the left sixth nucleus and the right third nerve, or innervation of the right internal rectus, may have been interrupted, but there is still the uninjured sixth nucleus, so that movement of the left eye outwards might have been expected to be unaffected. This was not so, and the facts suggest that conjugate deviation, being a voluntary act, the path connecting the nucleus with the higher centres may be the posterior longitudinal bundle, and that even with a sound sixth nucleus interruption of these fibres may result in such a paralysis. So that it may be regarded as possible that a lesion involving division of the posterior longitudinal bundle on both sides would result in paralysis of conjugate deviation on both sides, even though the sixth nuclei were unaffected.

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[1] Mott, "Experimental Enquiry upon the Afferent Tracts of the Central Nervous System." BRAIN, vol. xviii., 1895.

[2] Ferrier and Turner, "Cerebro-Cortical Afferent and Efferent Tracts." *Phil. Trans.*, 1897, not published at the time of writing this article.

It is generally believed that the sixth nucleus influences the opposite internal rectus indirectly through the cells of the third nucleus.[1] The present case, however, shows the existence of a certain number of degenerated fibres in the actual roots of the third nerves, which must have been derived from the posterior longitudinal bundles. The discovery of similar degenerated fibres in the fourth nucleus and nerve suggests some association in action between the internal and external recti and the superior oblique.

The situation of the lesion is such that the *seventh* nerve is destroyed at its loop, that is a point at which it would receive the fibres from the third nucleus to innervate the oculo-facial group,[2] and also those supposed to join it from a lower nucleus, ? the twelfth, to supply the lip muscles. As might be expected, the paralysis of the right face was fairly complete, resembling in fact an ordinary facial paralysis. The *nucleus* seems to be intact, but its cells present a large amount of deeply staining fatty granules, more so than on the left side—an example of the "*réaction à distance*" of Marinesco.

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[1] Gowers, "Diseases of Nervous System," 1893, second edition, p. 187.

[2] Tooth and Turner, *op. cit.*, p. 490, confirming a former observation by Mendel.

There are undoubted and numerous degenerated fibres in the *left seventh* trunk (fig. 14), which would lead one to expect a certain amount of paresis of the left face. Nothing, however, was noticed wrong with the left facial muscles, but slight weakness of them would have been hard to make out, because the paralysis on the right side would deprive the observer of his standard of comparison.

The eighth nerves and nuclei were unaffected on either side. The double degeneration of the white matter of the flocculi does not appear to have given rise to any symptoms.

A deviation of the tongue to the right, *i.e.*, away from the paralysed side, was noted, but passed off ten days before death. This may have been also a pressure symptom only, the *twelfth* nuclei and pyramidal tracts (except for a very few fibres above mentioned) being practically natural.

SUMMARY.

The following conclusions seem to be warranted on consideration of the foregoing observations.

1. *Lemniscus.*

(*a*) *Constitution.*—Consists of fibres which ascend only, the lowest origin being the nucleus cuneatus and n. gracilis.[1] The lemniscus rapidly increases in size in its progress brainwards, owing, no doubt, to a great accession of fibres from the sensory nuclei of the medulla.

(*b*) *Destination.* — As the corpora quadrigemina are approached the lemniscus gives off a dorsal branch, the lateral lemniscus, which appears to end in the corpora quadrigemina. The main or mesial lemniscus proceeds brainwards towards the optic thalamus, and here again appears to divide into two bundles, one of which loses itself in the optic thalamus; the other mass of fibres appears to take a lateral direction dorsally situated to the corpus subthalamicum. These fibres may enter the optic thalamus higher up, or they may pursue an uninterrupted course to the cortex.

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[1] Mott, *op. cit.*, p. 16.

(*c*) *Function.*—Its course and mode of degeneration suggest the transmission of afferent impulses[1] without doubt, but it can scarcely be the only path in the medulla and pons of afferent impressions. Its origin from the terminal nuclei of the two posterior columns of the cord suggest similar functions to those columns.

2. *Posterior Longitudinal Bundles.*

Consist of fibres which run to and from the brain in probably nearly equal proportions.

(*a*) *Descending fibres* appear to serve as a path of communication between the motor nuclei above and below the lesion. They can be traced to the anterior ground bundles of the lower medulla and upper cord, and in fact seem to be the main constituents of these bundles. The position of these fibres suggests a further connection between nuclei above and the anterior and lateral horns of the upper cord, including therefore the nucleus of origin of another cranial nerve, the spinal accessory. A certain homology between the "mixed zone" of the cord and the posterior longitudinal bundle seems probable.

(*b*) *Ascending fibres* enter into close relations with the cells of other motor nuclei (fourth and third). Fibres can also be traced into the roots of the fourth nerve on the same side and into those of the third (? on the opposite side). There is a free decussation across the middle line at the level of the third nucleus. Though most of the posterior longitudinal fibres seem to be absorbed in the third, yet a considerable number can be seen above it, and these probably enter into the optic thalamus.

3. *Flocculus of the Cerebellum.*

Its white matter consists largely of fibres derived from some higher level, probably the transverse fibres of the formatio reticularis.

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[1] It must, however, be remembered that Kingdon and Russell describe a degeneration of the fillet apparently secondary to extensive cortical degeneration in children, *Med. Chi. Trans.*, vol. lxxx., 1897, p. 109.

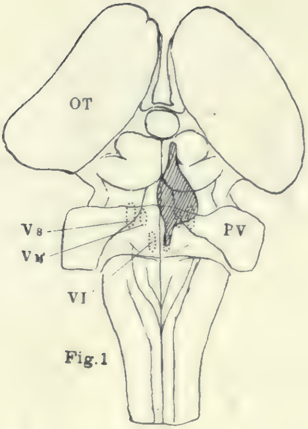

Fig.1

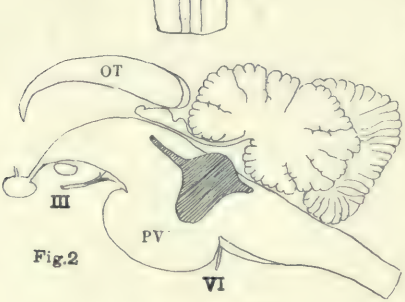

Fig.2

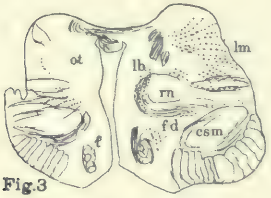

Fig.3

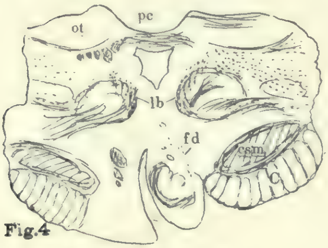

Fig.4

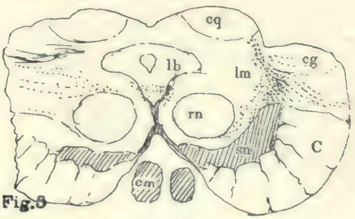

Fig.5

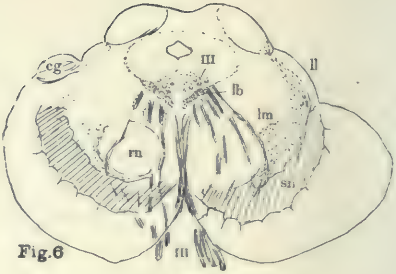

Fig.6

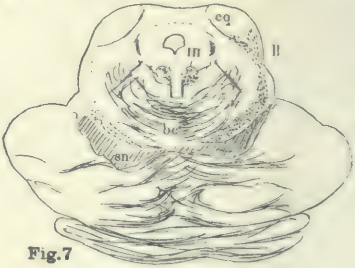

Fig.7

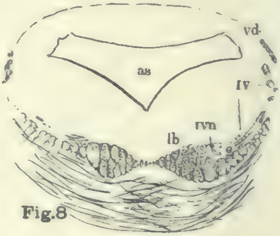

Fig.8

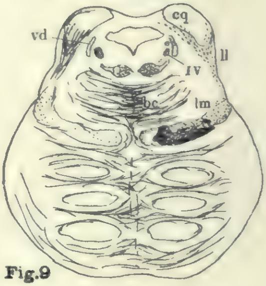

Fig.9

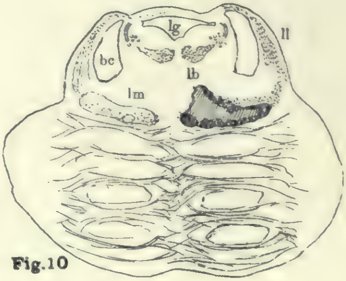

Fig.10

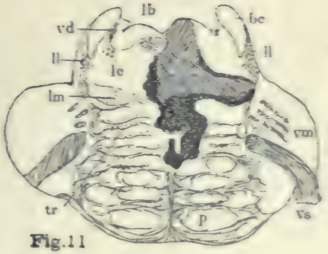

Fig.11

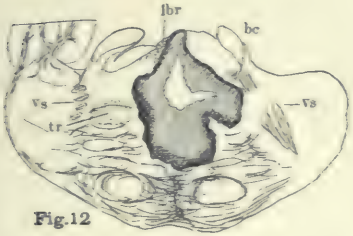

Fig.12

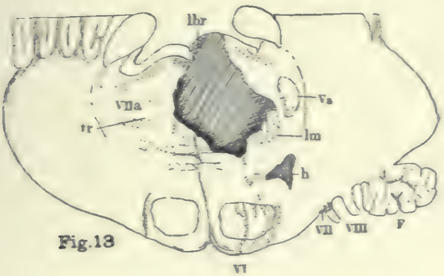

Fig.13

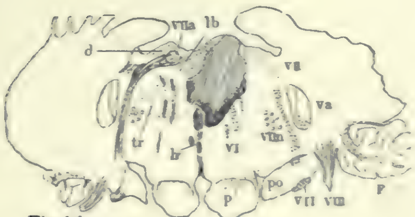

Fig.14

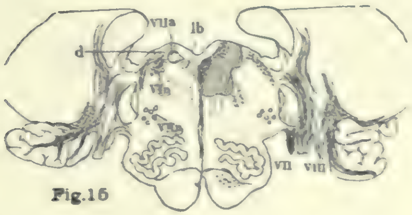

Fig.15

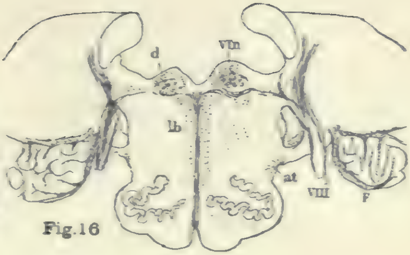

Fig.16

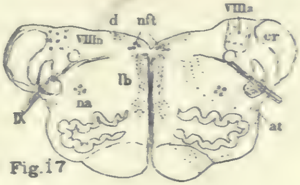

Fig.17

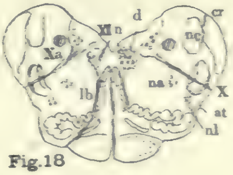

Fig.18

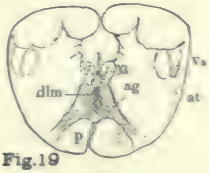

Fig.19

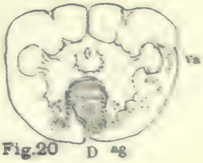

Fig.20

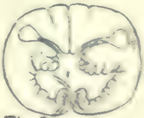

Fig.21

DESCRIPTION OF FIGURES.

FIG. 1.—Hæmorrhage viewed from above. *OT*, optic thalamus; *PV*, pons; *Vs*, sensory, *Vm*, motor nucleus of fifth; *VI*, sixth nucleus.

FIG. 2.—Side view of hæmorrhage.

FIG. 3.—Section (laterally oblique so that left half is at a higher level than right) at about the most posterior part of optic thalamus (*OT*) and third ventricle; *lm*, lemniscus; *f*, pillars of fornix; *fd*, a few degenerated fibres apparently belonging to the posterior longitudinal bundle (*lb*); *rn*, red nucleus; *csm*, corpus subthalamicum with crus ventral to it.

FIG. 4.—A little below the last. *c*, crus cerebri. (Other letters as above.)

FIG. 5.—At highest level of third nucleus on left side; *cq*, corpus quadrigeminum; *cg*, corpus geniculatum internum; *cm*, corpus mammillare; *sn*, substantia nigra.

FIG. 6.—Through the roots of the third nerves. *III*, third nuclei represented by little circles, the dots indicating degenerating fibres decussating; *ll*, lateral lemniscus.

FIG. 7.—Through the lowest part of the third nuclei; *bc*, brachium conjunctivum.

FIG. 8.—Enlarged diagram of central grey tube at level of fourth nucleus (*IVn*) showing its close relation with posterior longitudinal bundle (*lb*) with degenerating fibres in the issuing fourth nerve (*IV*); *as*, aqueduct of Sylvius; *Vd*, descending root of fifth.

FIG. 9.—Through pons at level of the posterior corpora quadrigemina (*cq*). Hæmorrhage on right side lying in totally degenerated lemniscus. Left lemniscus only slightly degenerated.

FIG. 10.—Below the corpora quadrigemina. Lateral lemniscus totally degenerated on right side, less so on left side passing outside the brachium conjunctivum and apparently sending a few fibres across in the lingula (*lg*).

FIG. 11.—At level of fifth nerves. *Vs*, sensory, and *Vm*, motor, about mid pons; motor roots of fifth totally degenerated owing to destruction of nucleus lower down; *lc*, locus cæruleus; *p*, pyramidal tract; *tr*, transverse fibres of substantia reticularis extensively degenerated. The deeply shaded part of the hæmorrhage represents a probably more recent effusion of blood.

FIG. 12.—A little below the last through the largest diameter of the hæmorrhage. *lbr*, right posterior longitudinal bundle totally destroyed. Left considerably degenerated.

FIG. 13.—At the upper level of the ascending loop of the seventh. *Va*, "ascending root" of fifth slightly degenerated in its ventral third; *VI*, right totally degenerated; *h*, small hæmorrhage; *F*, flocculus.

FIG. 14.—At level of seventh and sixth nerves, totally degenerated on the right side. Slightly also seventh derived probably from posterior longitudinal bundle, and from degenerated fibres which lie in the grey matter dorsal to it (*d*). *h*, small hæmorrhages; *VIIa*, nucleus of seventh; *p*, pyramid with a few degenerated fibres in it.

FIG. 15.—At level of upper end of olive.

Fig. 16.—Below hæmorrhage at level of lower part of sixth nuclei (*VIn*). Posterior longitudinal bundle fibres taking a vertical direction; *at*, a degeneration of descending fibres in the antero-lateral tract.

Fig. 17.—At level of ninth nerve. *VIIIn*, eighth nucleus; *VIIIa*, ascending root of eighth; *nft*, nucleus funiculi teretis; *cr*, corpus restiforme; *na*, nucleus ambiguus.

Fig. 18.—At level of upper part of twelfth nucleus (*XIIn*) and nerves, posterior longitudinal bundles lying inside the twelfth roots. *nc*, nucleus caudatus; *nl*, nucleus lateralis; *IXa*, ascending root of ninth.

Fig. 19.—Lower part of twelfth nucleus. Posterior longitudinal bundle continued as anterior ground bundle (*ag*); *dlm*, decussation of lemniscus undegenerated.

Fig. 20.—At decussation of pyramids. *D*, degenerated fibres of anterior ground bundles spreading round the anterior horns into the anterior root zones. Degeneration also of ascending root of fifth (*Va*), as before.

Fig. 21.—About level of first cervical. Anterior ground bundles still contain many degenerated fibres.

A STUDY OF THE LESIONS IN A CASE OF TRAUMA OF THE CERVICAL REGION OF THE SPINAL CORD SIMULATING SYRINGOMYELIA.

BY JAMES HENDRIE LLOYD, A.M., M.D.

Neurologist to the Philadelphia Hospital, Philadelphia.

IN 1894 I published a report of two cases of traumatic affections of the cervical region of the spinal cord simulating syringomyelia.[1] In both of these cases there had been severe injury, involving a fracture of the spine in the cervical region, and evidently a wound to the spinal cord. Both patients had survived for a long term of years after the original accidents, and both presented to a marked degree the clinical aspects of syringomyelia. Thus there was progressive muscular atrophy in the shoulders, arms and hands; spastic paraplegia with exaggerated knee jerks, but without persisting paralysis of the bladder and rectum; some trophic lesions, and, finally, the peculiar dissociation symptom, *i.e.*, preservation of tactile sense with abolition of sensibility to heat, cold and pain. These cases were regarded at the time as instances possibly of pachymeningitis cervicalis hypertrophica, although I distinctly stated that I did not intend to commit myself positively to a statement that they were necessarily or even probably examples of an inflammatory process. They simply conformed to the clinical type of syringomyelia, and I was not satisfied in my mind that they were not degenerative or necrotic rather than inflammatory.

Since the report of these cases one of the patients has died, and the autopsy and microscopic examinations are of such great interest that I desire to embody a report of them in another paper. The case has importance from two standpoints. First, with reference to the etiology of syringomyelia. Second, with reference to the light which it throws upon sensory conduction in the spinal cord. These subjects will be discussed after a description of the case and the sections.

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[1] *Journal of Mental and Nervous Diseases*, June, 1894.

In reporting the clinical history of the case here I shall abstract briefly the report in the former paper just referred to.

Patrick C., aged 60 years, white, male, labourer, had received when 30 years of age an injury to the cervical and dorsal spine by being jammed between two canal boats in a canal lock. For treatment he said that he was kept strapped to a long dorsal splint or platform for five weeks. He must have made a good recovery, for in about three months he returned to work as a common labourer, and continued to do hard work up to the time when he received a second severe accident. This second accident happened about two years before his admission to the hospital, hence after an interval of nearly thirty years. He was in good health at the time. While digging out a clay bank he was buried under a landslide, where he remained about one half hour before he was rescued. He was unconscious for twenty-four hours. On recovering consciousness it was found that he had paralysis in the left arm and leg. The face was not involved and there was no affection of speech. He remained in the hospital for six months. Contractures gradually began in the affected arm and leg.

His condition when first seen by me, three years after the accident, was as follows:

There was an angular deformity, involving the seventh cervical and first and second dorsal vertebræ, and there was a depression also in the region of the lumbar vertebræ. The deformity caused the patient to carry his head in a set position, his chin almost resting on the thorax (fig. 1). The left arm and leg were paralysed, and there was contracture of the arm and forearm. The shoulder muscles and muscles of the arm were paralysed and atrophied, and there were fibrillary contractions in them. Clonus was marked in the fingers, wrist, and ankle of both sides, and the phenomena of so-called spinal epilepsy were readily obtained in the legs. This general leg clonus once started would continue almost indefinitely.

The sensory symptoms were of great interest. The patient was unable to distinguish heat from cold or to recognise either in the region marked on the chart. It is to be observed that this thermo-anæsthesia was on the right side or the side opposite the

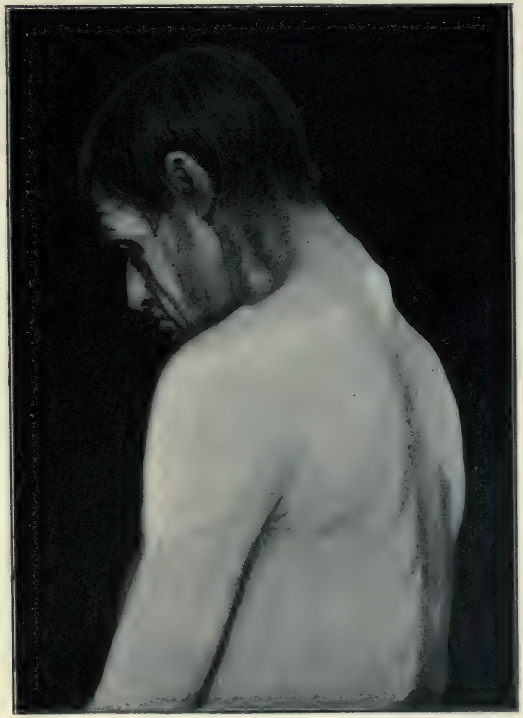

FIG. 1.

A case of trauma of the cervical region of the spinal cord.

paralysis of motion (figs. 2 and 3). There was one area of anæsthesia on the right leg (fig. 4). There was also a form of hyperæsthesia or rather paræsthesia of the right or unparalysed side. Thus he experienced and complained much of a sensation of burning on this side, and wanted constantly to put cold water on it to allay the heat, but he found this remedy ineffectual.

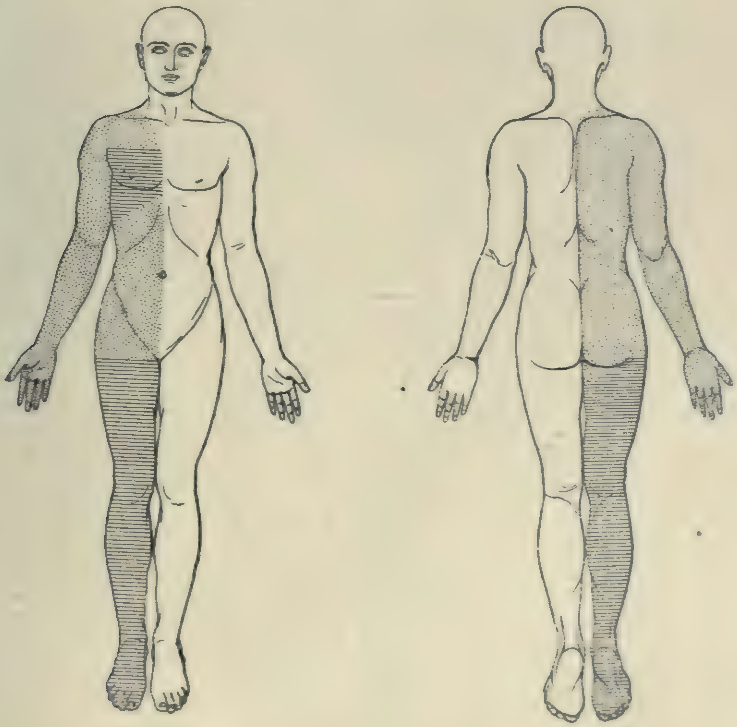

FIG. 2. FIG. 3.

Thermo-anæsthesia indicated by cross-lines. Analgesia in the same areas, and also in areas indicated by dotted shading.

Finally, he was profoundly analgesic in the right arm, side and leg. The iris was not involved, nor was there any impairment of respiration or any interference with the innervation of the bladder.

Commenting on this case in the former report I observed that its prominent feature was its hemiplegic type. This was so pronounced that the case closely resembled one of cerebral lesion. That it was not such, however, was clear from its history, as well as from the deformity of the spine, the absence of paralysis of the face or tongue, the sensory changes and the atrophy and fibrillation in the paralysed muscles. It is to be observed that it followed closely the Brown-Sequard type, *i.e.*, the paralysis of motion was on one side and the sensory changes on the other. Its resemblance to syringomyelia was borne out by the peculiar dissociation symptom and the muscular atrophy in the shoulder and arm. As to its hemiplegic type, it must be borne in mind that syringomyelia occasionally presents this

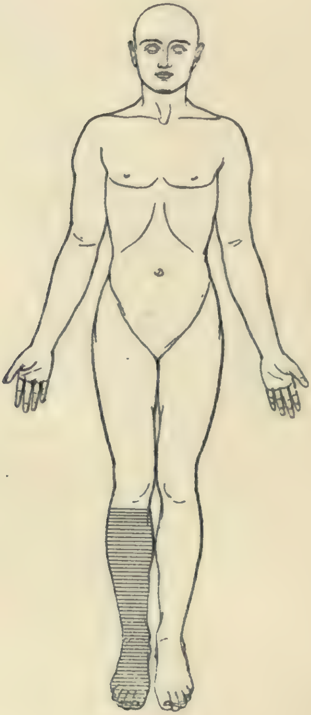

FIG. 4.

Tactile anæsthesia, varied with delayed sensation.

form. That it was the direct effect of accident was evident from the fact that it supervened immediately on the receipt of the injury from the clay bank, which had evidently resulted in a fracture of one or more of the vertebræ of the lower cervical and upper dorsal spine. It seems certain that the injury was the result of the second accident, because the man had completely recovered from the effect of the first accident thirty years before, so that he had been able to do hard work.

The patient died about one year ago from some intercurrent affection. Thus an interval of about five years had elapsed between the receipt of his second injury and his death.

The findings at the autopsy were as follows :

Before the spinal canal was opened, a deformity of the 3rd, 4th, and 5th cervical vertebræ was seen. The spinous processes of these vertebræ were gone, and an osseous mass, resembling callus, was seen on either side of the spinal column. The cord was removed quite easily up to the 6th cervical vertebræ, where the dura was adherent anteriorly to the body of the vertebra, but was free posteriorly. At the junction of the 4th and 5th cervical vertebræ an angular deformity, projecting into the spinal canal, compressed the cord, which was here ribbon-like. The point of greatest compression was at this prominence. The bodies of the 3rd and 4th cervical vertebræ were flattened. Again, at the 2nd and 3rd cervical vertebræ there was an angular projection, caused by a fracture and dislocation of these two vertebræ, with, however, no corresponding deformity in the spinal canal. On the body of the 7th cervical vertebra there was a sharp projecting point. It seemed to be an exostosis, but there was no other evidence of fracture in this region. The cord was much flattened antero-posteriorly at the exits of the 5th, 6th and 7th cervical roots. At and above the exit of the 4th cervical roots the cord was little if any deformed. At the exit of the 8th cervical roots the cord began to assume a normal appearance, and nothing abnormal was noted below this level. The dura at the 3rd and 4th roots was fully twice as thick as normal at the posterior part, but anteriorly it was normal. There was no other distinct evidence of meningitis, unless it was at the 5th, 6th and 7th posterior roots and on the left side only, where the arachnoid was a little more adherent than is usually observed. There was a spiculum of bone attached exactly to the median and lateral parts of the dura at the exit of the 7th roots, and at this point there was a mass of fibrous tissue possibly resulting from external pachymeningitis. In the brain nothing abnormal was noted.

MICROSCOPIC APPEARANCES.

Seventh Cervical Segment (fig. 5).—This is the level of greatest destruction. The whole cord is flattened or ribbon-like at this point. The left half of the cord is extensively injured. The area of greatest involvement includes the left antero-lateral column (the pyramidal tract, cerebellar tract, and Gowers' tract), the anterior and posterior horns, the anterior portion of the posterior columns, and the gray and the white commissures. The lesion extends across to the right side of the cord along the commissure, but in its greatest intensity stops before reaching the horns of the right side. The right half of the cord, however, shows some diffused degeneration of slight degree in the antero-lateral column, and the anterior and posterior horns are misshapen and slightly degenerated. It is interesting to note that the lesion occupies much the same ground as most cases of syringomyelia, also that the posterior columns are affected chiefly in their anterior part, *i.e.*, contiguous to the gray commissure. The left half of the cord in the lateral column is a mass of necrosed tissue, including everything except the extreme anterior column and column of Türck, which are well preserved. The affected area of the posterior columns at this level does not include Schultze's comma zones. It is a somewhat irregular mass of degenerated tissue beginning at the gray commissure anteriorly and extending in a posterior direction so as to include not quite one-third of the columns. In a lateral direction it extends

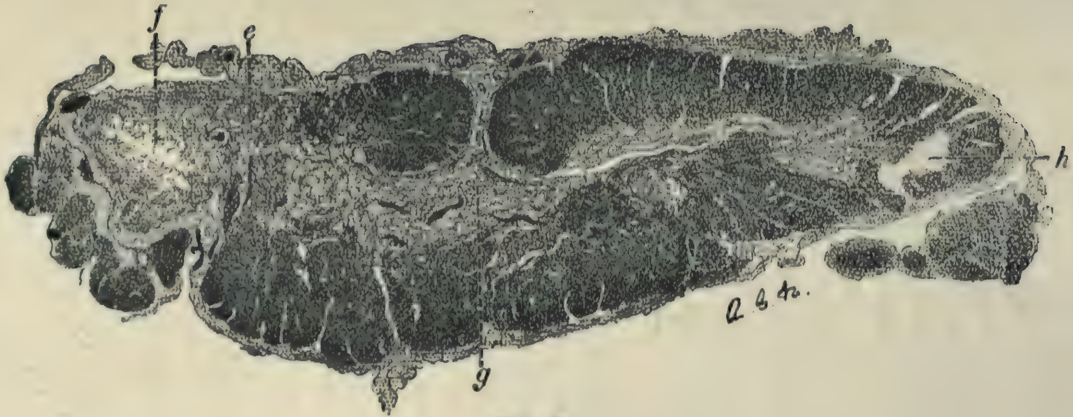

FIG. 5.

Seventh cervical segment.

The section represents the area of greatest degeneration.

(*e*) Degenerated left anterior root.
(*f*) Degenerated lateral column.
(*g*) Degenerated zone about the central canal.
(*h*) Slight area of degeneration in the right lateral column, which cannot be traced very far downward.

irregularly as far as the posterior horns and is rather more extensive in the left posterior column than in the right. It has no definite outline, and cannot be said to be limited to any special group of fibres. It is, however, the starting place for an ascending degeneration in the posterior columns which at higher levels assumes a peculiar branching or W form which will be referred to later.[1] It is thus seen that a large portion of the fibres in the posterior columns escape the lesion (and this doubtless accounts for the preservation of tactile sense).

There is no cavity formation in any part of the cord at this level, nor is any cavity found at any other level of the cord. It is to be noted, however, that the position of the lesion is practically identical with what is seen in many cases of syringomyelia. Thus there is a well marked degenerative zone about the central canal involving both the gray and the white commissures, which at this level are quite obliterated. The only degeneration in the posterior column is that which is contiguous to this central zone of necrosed tissue. This extensive degeneration of the central gray matter of the cord must have acted effectually as a bar to the transmission at this level of any impulses through them from one side to the other. Finally, the extensive necrotic area on the left side, involving both the posterior and anterior horn, the pyramidal tract, the direct cerebellar tract, and Gowers' tract, acts almost though not quite as a hemisection of the cord. The only notable fibres to escape are those in the posterior columns and in the anterior columns and columns of Türck.

Starting from this point we may first trace the ascending degeneration through the higher levels of the cord and, second, the descending degeneration below the seat of the lesion.

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[1] Bruce ("On the Endogenous or Intrinsic Fibres in the Lumbo-Sacral Cord," BRAIN, part lxxix., 1897), refers to the cornu-commissural tract in the lumbar region, described by Marie as being composed of endogenous fibres. This tract lies in the anterior part of the posterior column in close apposition to the posterior horn, commissure and septum. It does not degenerate in locomotor ataxia, or after injuries to the cauda equina, but degenerates after injuries to the posterior horns, which are supposed to contain its cells of origin. It also degenerates in syringomyelia. This degeneration is said to be descending. It is almost this identical area that has degenerated in my case, only in this instance the tract is in the cervical region, and the degeneration appears to be upward. That these fibres are endogenous is highly probable.

Sixth Cervical Segment.—At this level the left half of the cord is also much involved. The degenerated area involves the left pyramidal tract, cerebellar tract and Gowers' tract, and the left posterior and anterior horn. The posterior columns are only involved in the region contiguous to the gray commissure. Hence most of the fibres of the posterior columns escape. The left column is rather more involved than the right. It is to be noted that at this level both the grey and white commissures preserve their normal size, shape, and appearance. There is no dilatation or cavity formation.

Fifth Cervical Segment.—The cord here is still misshapen. The left lateral region is smaller and deeply degenerated. The degeneration in the posterior columns still retains the general outline that was seen at the lower level. The anterior horns are normal in shape and size, but possibly have fewer multipolar cells than normal. The right pyramidal tract and direct cerebellar tract are the seat of a very slight diffuse degeneration. This is seen especially in the pyramidal tract.

Third Cervical Segment.—The posterior columns still present some areas of ascending degeneration. These areas have now taken a more definite shape. They are branching streaks of degeneration passing outward or backward from the region of the gray commissure. The great mass of fibres, however, in the posterior columns are normal. On the left side the cord is still slightly deformed, and here the cerebellar tract and Gowers' tract are deeply degenerated and the crossed pyramidal tract is also the seat of diffuse degeneration. (Ascending degeneration of the pyramidal tract). The central region of the cord (posterior gray and anterior white commissure) is normal. The anterior horns appear at this level to have some degenerated multipolar cells. The right horn especially is smaller than the left, and has not many large cells.

Upper Cervical Cord.—The same general appearances are seen as in the third cervical segment, except that the anterior horns at this level are normal and contain many large multipolar cells.

Decussation of the Pyramids.—At this level the posterior columns show very plainly two well defined areas of degeneration forming lines or streaks extending first from near the grey commissure in a posterior direction through Burdach's column, and second from a point in Goll's column at the posterior longitudinal fissure backward and outward not extending to the border of the cord. Neither of these zones occupies exactly the region of Schultze's comma zone. It is thus seen, as said already, that the posterior columns are not degenerated in their entirety, but that the vast mass of their fibres escape. It is difficult to account for these peculiarly shaped areas of degeneration in the posterior columns. They maintain the same shape through several segments of the cord above the level of injury. The question may be raised whether these areas of degeneration are caused by the arrangement of the fibres coming in at the point of injury, passing gradually inward toward Goll's column as they ascend. It is possible, however, that they are endogenous fibres, originating in the gray matter at the seat of injury. The cerebellar tract and Gowers' tract are deeply degenerated on the left side.

Medulla Oblongata, Decussation of the Lemniscus.—At this point the direct cerebellar tract and Gowers' tract are still deeply degenerated on the left side. On the right side they are normal. The antero-lateral ground bundles are normal. The nucleus gracilis and nucleus cuniati as compared with sections of normal cords are normal. The posterior columns at this level do not appear degenerated, certainly Burdach's column is not. Goll's columns show deeply stained (non-degenerated) rim of medullated fibres around the nuclei gracilis. (This has been carefully compared with selections from a normal medulla with the result that there may be slight evidence of degeneration in Goll's columns ; if so, this probably represents the group of degenerated fibres running upward through the posterior columns as observed at lower levels.) At this level some of these fibres have probably already terminated in the nuclei gracilis and cuniati. It is thus of interest to note that at this the highest level of the posterior columns their great mass of fibres is not degenerated. There is no evidence of ascending degeneration of the pyramidal tract beyond one or two segments above the seat of injury. This is in marked contrast with what I found in a case of syringomyelia formerly reported,[1] in which there was deep ascending degeneration of one of the pyramidal tracts extending through and above the decussation of the pyramids.

Descending Degeneration.

Eighth Cervical Segment (fig. 6).—This is just below the seat of injury. The cord is slightly contracted in form on the left side. In the posterior columns Schultze's comma zones are plainly degenerated (descending degeneration). There is also slight diffuse degeneration in the posterior columns which extends only a short distance downward. This is probably caused by the degeneration of the descending branches of the exogenous fibres. It is rather too plainly marked in the illustration. The left lateral pyramidal tract is deeply degenerated, the right only slightly so. The anterior horns contain many multipolar cells some of which are possibly somewhat degenerated. The direct cerebellar tract and Gowers' tract are normal on both sides.

First Dorsal Segment.—The crossed pyramidal tract on the left side is deeply degenerated. This tract on the right side is only slightly degenerated. In the posterior columns Schultze's comma zones are still degenerated. There is also some diffuse degeneration in these columns already referred to (descending branches of exogenous fibres). The anterior horns contain normal multipolar cells.

Second Dorsal Segment. — Deep degeneration of the left lateral pyramidal tract. The slight degeneration in the posterior columns is not so marked as in the preceding section. Schultze's comma zones are not so distinct. Horns normal. From this level downward the cord presents simply deep degeneration in the left lateral pyramidal tract. The posterior columns soon become normal in their entire extent.

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[1] *University Medical Magazine*, March, 1893.

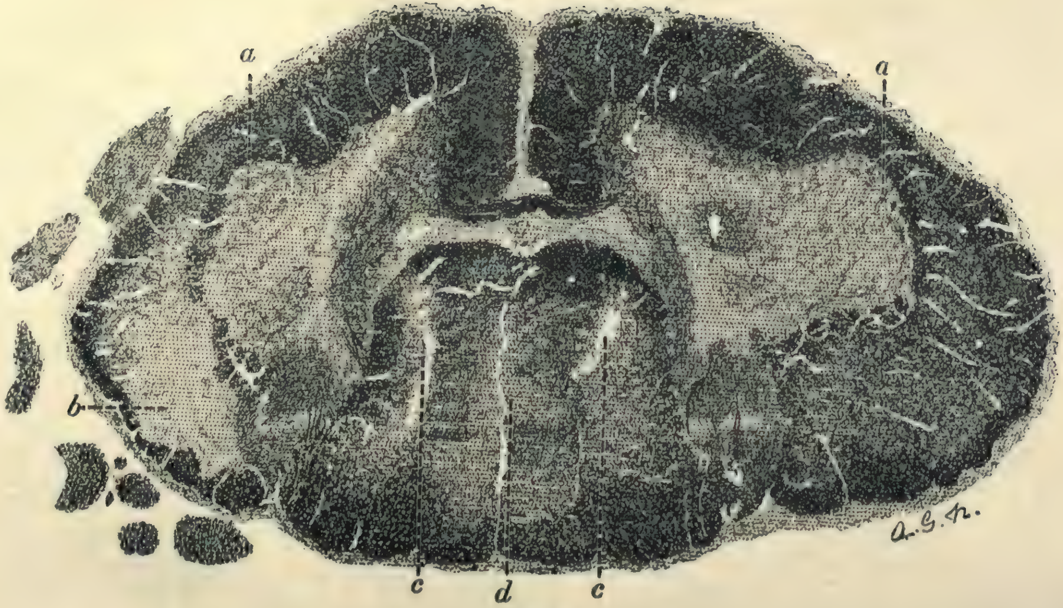

FIG. 6.

Eighth cervical segment.

(*aa*) Anterior horns containing fewer fibres than normal.
(*b*) Degenerated left lateral column.
(*cc*) Degenerated comma zones of Schultze.
(*d*) Slight degeneration of posterior columns which extends only a short distance downwards.

COMMENTS.

With reference to the etiology of syringomyelia this case may possibly have some significance. It is true that no cavity had formed in this cord, and the case therefore was not one of true syringomyelia. The seat of the lesion, however, especially about the central canal and in the central grey matter, was practically identical with that of many cases of syringomyelia. So true is this that in looking at these sections the observer feels that he is studying a lesion which only required to advance a step further to become truly a case of cavity in the cord. It would seem that it must have been comparatively easy for this necrotic tissue to have broken down still further and to have been absorbed, thus leaving a cavity of irregular extent and outline in the central region of the cord. If this had occurred in the present case the cavity would have occupied especially the region of the grey commissure extending into the posterior column of the left side, and possibly including the lateral column also. For some reason cavities in the cord, as we know, form most readily in this very region of the grey commissure. The causation of them in most cases is obscure, but the possibility of their being caused by trauma has long been recognised. The interesting question arises, therefore, in this case, whether we are not looking at the result of a trauma which has produced an area of necrosed tissue in this usual place without having advanced to the final step of cavity formation. While this question is somewhat speculative the appearances in this cord are highly suggestive of such a state. Cases are not wanting which show the action of trauma in causing cavities in the central grey matter of the spinal cord. Silcock[1] reported a case of fracture of the fifth cervical vertebra, with injury to the spinal cord, in a man who had dived into shallow water. In this case the patient lived for some months. At the autopsy a cavity was found lying immediately inside and parallel to the right posterior horn. There was also much general sclerosis and disorganisation. Among others who have observed trauma as a cause of syringomyelia are Harckin, Strümpell, and Oppenheim, quoted by Bruhl.[1] They have especially noted falls from high places.

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[1] *Trans. Path. Soc. Lon.*, vol. xxxix., 1888, p. 18.

According to Hinsdale[2] the syndrome of syringomyelia is likely to occur suddenly in traumatic cases, and we may then suspect the existence of hæmatomyelia as a starting point for the cavity formation. Hæmorrhages of the spinal cord, as is well known, affect chiefly the central grey matter. According to this same author, Minor observed the symptoms of syringomyelia in organic lesions of the spinal cord resulting from trauma. While it is true that hæmatomyelia may be the starting point for cavity formation in the cord in most cases due to trauma it does not follow that this condition must be the only one. It is supposable at least that necrosis and softening might occur without hæmorrhage and eventually produce a cavity. In the present case there is no evidence of a hæmorrhagic cyst in the cord. All that can be said is that a necrotic area has been produced in the region most favourable for the production of a cavity, and that, clinically, the case had close resemblance to one of syringomyelia. Other cases might be quoted from the extensive literature of this subject.[3]

The question of sensory conduction in the spinal cord is still one of some obscurity. So far as one pathological case can throw light upon this subject some inferences may possibly be drawn from the case under consideration. In the first place is to be noted that tactile sensibility was not involved, except in a limited area on the right lower extremity. Why this comparatively limited and isolated area was involved does not appear plain from the microscopic appearances. It is to be observed, however, that this tactile anæsthesia preserved the shape and extent of the ordinary segmental anæsthesia so commonly seen in hysteria. Thus it involved the whole of the foot and leg as high as the knee where it

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[1] "Contribution a l'étude de la Syringomyélie," Bruhl, Paris, 1890.

[2] "Syringomyelia," Alvarenga Prize Essay, College of Physicians of Philadelphia, 1897.

[3] For an extensive bibliography, including more than five hundred references, Hinsdale's Prize Essay, already quoted, may be consulted.

was sharply limited by a line drawn horizontal to the long diameter of the limb. In short, it had the appearance of a stocking, or, as when seen in the arm, of a gauntlet. This segmental anæsthesia, not following the distribution of any particular nerve, is hard to explain by an organic lesion. In considering its significance in this case I have been strongly inclined to regard it as an hysterical phenomenon. The patient had some of the mental characteristics of hysteria, and it may well be that this was one of the hysterical stigmata that not uncommonly complicate the picture of organic nervous disease. That it was not due to organic changes in the posterior columns of the cord is rendered probable by the fact that the columns of Goll at and above the seat of lesion were not seriously involved. Moreover, although slightly implicated, they were equally so on both sides. We know that the sensory fibres from the lower limbs pass upward through the cervical region exclusively in these columns of Goll. Therefore this case presents no reason why total tactile anæsthesia of this peculiar segmental type should be found in only one leg. In the second place, this patient had the peculiar disassociation symptoms of syringomyelia involving the whole right half of the body. The thermo anæsthesia was not so extensive as the analgesia, since the former involved only the lower limbs and a limited region on the thorax. Finally it is to be observed that this hemianæsthesia to pain and temperature was on the side *opposite* to the main lesion in the cord and to the motor paralysis. Hence in a sense it presents the appearance—without tactile anæsthesia, however—of a Brown-Sequard paralysis of the spinal cord.

It seems that it is not difficult in the present case to explain the sensory phenomena by the lesions found under the microscope. In the first place, the preservation of tactile sense is perfectly explained by the fact that the posterior columns of the cord were not seriously involved. The parts of these columns most involved were contiguous to the posterior grey commissure, a region in which the endogenous fibres especially of the posterior columns run. The fibres passing in by the posterior roots (exogenous fibres) pass up the posterior columns, gradually seeking and entering the columns of Goll. As already said, the columns of Goll are not deeply degenerated at the level of the lesion nor above the level of the lesion. According to Van Gehuchten the probabilities are that these long fibres of the posterior columns, *i.e.*, the exogenous fibres, passing in by the posterior roots, serve for the conduction of tactile sensibility. That is the lesson taught by syringomyelia and also by the present case. These fibres pass up directly into the nuclei graciles and cuniati in the lower part of the medulla oblongata. Here their arborisations are distributed to the neurons of a second order. The microscope in this case does not indicate any marked degeneration of these fibres at or about their point of entrance into these nuclei.

With reference to the thermo anæsthesia and analgesia in this case, I may say that they seem to point unerringly to the conclusion of Van Gehuchten, who says that in all probability pain sense and thermic sense are transmitted through the grey matter by way of cells whose axis cylinders become the fibres which constitute especially the tract of Gowers. These fibres according to the same authority are *crossed*. In the present case Gowers' tract as well as the direct cerebellar tract was deeply degenerated on the *left* side from the seat of lesion all the way up. On the right side there was very slight evidence of degeneration in these two tracts. This would put the case in perfect harmony with the anatomical demonstrations of Van Gehuchten. The tract of Gowers on the left side above the lesion being degenerated and this tract being composed of axis cylinders arising from cells located in all the segments of the grey matter of the opposite side, it follows of course that the thermo anæsthesia and analgesia would be upon the opposite or right side. This was so in this case. The motor symptoms, *i.e.*, spastic paralysis of the arm and leg with muscular atrophy of the shoulder and upper extremity, were most marked in this case, as always, upon the side of the lesion, *i.e.*, the left side. If this view is found to be confirmed by other cases we must accept a modified view of Brown-Sequard's paralysis of the spinal cord. Instead of this paralysis being characterised by an hemianæsthesia on one side and a hemiplegia on the other we shall have the syndrome constituted as follows:—The motor paralysis and the tactile anæsthesia would be on the side of the lesion, while the anæsthesia to pain and temperature would be on the side opposite the lesion. Of course, as in all pathological cases, the present case is open to the objection that the lesion is not sharply defined nor limited strictly to the one side. Yet in looking without prejudice at the sections it must be acknowledged that the lesion is confined very largely to the left side.

It is but fair to say that all anatomists and physiologists do not as yet agree with Van Gehuchten that the tract of Gowers is composed of crossed fibres, or that it is necessarily the pathway for the pain and temperature sense. Yet the present case points strongly to some such conclusion.

According to Gotch[1] the degenerative changes which follow section of a posterior root are always limited to the side of the lesion and affect only the fibres of the posterior column. This degeneration ceases at the medulla oblongata in the grey matter of the gracile and cuneate nuclei. As the fibres entering the posterior roots divide dichotomously, one branch, already referred to, passing upward, and the other branch looping downward for a short distance to pass into the central grey matter, it follows that in a localised lesion of the posterior columns there might be found some slight descending degeneration in these columns for a short distance below the lesion. There is some evidence of this, but to a very limited extent downward, in this case. Gotch, however, contends that there is no evidence of ascending degeneration on the opposite side of the cord either after a section of a posterior root or after a hemisection. This, however, does not invalidate the claim made here, since the fibres in Gowers' tract and the direct cerebellar tract are *endogenous* fibres, *i.e.*, take their origin in cells located in the grey matter at the various levels of the cord itself. Impulses from painful and thermal impressions, according to this observer, radiate into the grey matter of the cord, and pass particularly into the lateral columns of the opposite side. This is perfectly in accord with our present case. Hence, to recapitulate briefly, it may be said that tactile impressions pass directly up the posterior columns by way of the exogenous fibres of the same side, while painful and thermal impressions pass into the grey matter and through the cell bodies of a second order of neurons whose axis cylinders, in a large majority at least, pass across to the opposite side of the cord and up the lateral columns, especially in Gowers' tract. This seems to be the teaching of the case here under review.

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[1] "Recent Research on the Spinal Cord," by Francis Gotch, M.A., F.R.S., *Liverpool Medical Chi. Journ.*, xiii., 1893, p. 150.

In conclusion, I have to express my obligation to Dr. William G. Spiller for his skilful preparation of the sections in this case, and to Miss Anna G. Newbold, of Philadelphia, for her very accurate drawings of the same.

GENERAL PARALYSIS OF THE INSANE DURING ADOLESCENCE, WITH NOTES OF THREE CASES.

BY PURVES STEWART, M.A., M.D.EDIN., M.R.C P.LOND.

Senior Resident Medical Officer, National Hospital, Queen Square, London ; late Assistant to the Professor of Medicine in the University of Edinburgh.

THE occurrence of general paralysis of the insane in patients about the age of puberty is now an acknowledged fact. Although somewhat uncommon, it is becoming better recognised, and within the last fifteen years a number of cases have been placed on record, gathered chiefly from amongst asylum patients. The three following cases, however, may be of interest, inasmuch as they occurred in the course of ordinary neurological work, and were therefore studied from the point of view of the neurologist rather than that of the alienist. They also help to throw light upon the possible causal antecedents of the disease in patients of so young an age, and they exemplify well its peculiar symptomatology in such patients, whilst one of the cases also presents some interesting points in the study of epileptiform fits.

For permission to publish my notes of these cases I am indebted to the courtesy of Dr. Hughlings Jackson, Dr. Ferrier, Dr. Beevor and Dr. James Taylor.

Case I.—W. H., male, aged 16$\frac{11}{12}$ years, was admitted to the National Hospital, Queen Square, on October 1, 1897, under the care of Dr. Hughlings Jackson. He was said to be suffering from "fits."

Family History.—None of patient's relatives have ever suffered from any nervous or mental disease. His father is alive and healthy; his mother died in childbirth at the age of 41. Patient is the second of fifteen children, of whom the first child was born dead and five others died at birth. The remaining members of the family, with the exception of the patient, are perfectly healthy, he being the only delicate member of the family.

Previous Health.—Although never robust, he has not suffered from any definite illnesses beyond the usual ailments of childhood. He never had any convulsions in infancy. There was no history of injury or accident of any sort.

Present Illness.—Patient was formerly a very smart, intelligent boy and very good at his lessons, but about three years ago, when he was 13 years of age, gradual mental dulness came on. The first observed symptom of this was that, when he was sent out on errands, he used to return having completely forgotten several of his messages. He became gradually much duller mentally, quieter and less lively.

About two years ago he had his first "fit." On getting up one morning he complained of feeling sick and went back to bed, when he became unconscious, with pale face and twitching of all his limbs. This twitching is said to have lasted a couple of hours. During the fit he did not bite his tongue, nor were the sphincters affected. After the fit he seemed mentally more peculiar for some days, with hallucinations of vision in the form of moths, &c. These hallucinations passed away in a few days.

Since then he has had three more fits—four fits in all—at intervals of about six months, the last fit occurring six weeks before his admission. For a day or two before each fit he has usually been "quiet and moping." During the fits he has never bitten his tongue, nor has he passed water. In two of the fits he has fallen down at the onset. After each fit for about ten days he has always been mentally peculiar, and his right arm is said to be weak—he "loses the use of it" for a day or two. Patient is naturally right-handed, but now his right hand is said to be much weaker than his left.

Ever since the onset of the illness his father has noticed the patient's gait to be somewhat staggering in character. The boy himself does not complain of giddiness, even when staggering.

Condition on Examination.—Patient is a pale-faced boy of fair muscular development, with sharply cut features. He has a vacant expression and his mouth hangs open. There are no signs of congenital syphilis in the teeth, eyes, face, or elsewhere.

There is considerable mental hebetude. He never volunteers any remark. He answers questions slowly and often incorrectly, speaking in a slow monotonous voice with marked fibrillary trembling of lips, face and eyelids. His memory is very defective. Although usually in a quiet, dreamy mental condition, he has occasional noisy bursts of passion in which he obstinately refuses to comply with simple requests.

Smell, taste, and hearing are normal. The visual fields are not contracted, visual acuity is normal, and the optic discs are healthy.

The pupils are widely dilated, the left more so than the right. They do not react to light, and there is very slight, if any, contraction during convergence. There is no squint, nor nystagmus.

The facial movements are normal save for the fibrillary tremor above noted. The palate rises normally on phonation. The tongue is protruded straight and is not atrophied, but it exhibits marked fibrillary tremors on both sides.

There is no anæsthesia anywhere on the face, trunk, or limbs to the lightest touches or pricks. All the movements of the upper limbs are performed, but in a languid manner, and the grasp of the right hand is slightly weaker than that of the left. In the lower limbs there is a similar languor in all movements, but no actual paralysis. Patient's gait is somewhat ataxic; he walks on a broad base, stamping his heels. He does not sway or fall with feet together and eyes closed. The knee-jerks are brisk and equal. There is no ankle clonus. Supinator and triceps jerks are brisk, and there is marked jaw-jerk.

The plantar, cremasteric and abdominal reflexes are normal and equal on both sides. The sphincters are normal, but owing to his mental dulness he does not always obey the calls of nature, frequently passing both urine and fæces as he walks about. He practises onanism with considerable frequency. The genital organs are much smaller than normal, and there is no pubic hair. The lungs, abdomen and urine are normal. There is a marked systolic murmur at the apex of the heart, conducted outwards to the left side.

The patient remained in the same dreamy mental condition for a month after his admission, with occasional attacks during which he was noisy and passionate. He never expressed exalted ideas of any sort.

On *November* 3rd patient was seized with epileptiform convulsions which came on during the night when he was asleep, and which lasted for nearly four hours without intermission. The fit commenced with a cry, and the twitching was observed to begin in the fingers of the right hand. Owing to the long duration of the fit I had an opportunity of studying its character with some minuteness. The patient, who was completely unconscious, lay on his back, with rapid clonic spasms of all the muscles of the limbs, trunk and face, the right side being distinctly more severely affected than the left. His face was natural in colour. The eyes were open but with occasional clonic spasms of the orbiculares palpebrarum. There was marked conjugate deviation of the eyes to the right side. The pupils were widely dilated, equal, and insensitive to light. The mouth was widely open. There were no twitchings of the jaw muscles, but the face twitched markedly, especially on the right side. The palate twitched markedly and symmetrically on the two sides. Laryngeal examination showed rapid alternate separation and approximation of the vocal cords, and also alternate narrowing and widening of the aryteno-epiglottidean folds.[1] There was also distinct twitching of the tongue on both sides. All the muscles of the limbs and trunk on both sides twitched briskly and synchronously with the face and tongue muscles. There was marked twitching of all the muscles above and below the hyoid bone, and of both sterno-mastoids equally. The head was drawn backwards and the chin elevated. Respiration was noisy and rendered irregular by the clonic spasms of the respiratory muscles. There was occasional inspiratory stridor. There was no twitching of the anus.

The clonic spasms of all these muscles were irregular in rhythm, the number of contractions counted in a minute being about 180. Patient's skin was warm and moist; the temperature 98·2° F., pulse 156, and the respirations 36 per minute. Patient did not pass urine during the fit, nor did he bite his tongue, owing to the jaws being widely open all the time.

During the fit the knee-jerks were elicited readily.

Next day, *November* 4th, even after large doses of chloral and potassium bromide *per rectum*, although the general convulsions had ceased, twitching still persisted in the left side of the face and tongue and in the left hand; with this there were occasional spasms of tonic rigidity in the trunk and non-twitching limbs. Patient was still unconscious; the eyes were blood-shot and directed upwards and to the right side. The knee-jerks were present and equal, not exaggerated. There was no ankle clonus.

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[1] The vocal cords have not hitherto, I believe, been observed during an epileptic fit in the human subject. Analogous observations on the larynx have, however, been made by Risien Russell during absinthe convulsions in monkeys, and by Semon and Horsley in excitation of the cerebral cortex. "We have observed," they say, "that in powerful or long-continued excitation of the laryngeal area, true epilepsy of the vocal cords supervenes, gradually spreading to the neighbouring muscles of the face, neck, head and upper limbs. From this we conclude that the epileptic cry is not, as has been so long believed, the result of a discharge primarily from the medulla oblongata, but rather represents a stage in the cortical excitement popularly termed an 'epileptic fit.'" (Semon and Horsley, "On the Relation of the Larynx to the Motor Nervous System," *Deutsche Medicin. Wochenschr.*, 1890, No. 31, p. 10.)

November 7th.—Patient was somewhat brighter; semi-conscious, but did not speak; became able to swallow again. Occasional twitchings still occurred at intervals in the face alone, sometimes on one side, sometimes on the other.

On *November* 8th patient again became more deeply comatose; twitching entirely ceased. The pupils were dilated and insensitive. Cheyne-Stokes respiration set in, and he died from respiratory failure.

The *post-mortem* examination was made by Dr. Risien Russell, who made the following report :—

Autopsy.—A *post-mortem* examination was made eighteen and a-half hours after death. The skull-cap was natural, as was the dura mater; no undue adhesions existed between these two structures, nor was the dura mater thickened or anywhere adherent to the pia-arachnoid. There was slight but distinct general thickening of the lepto-meninges over the convexity of both cerebral hemispheres, where they presented a milky, opaque appearance. So adherent were the meninges to the surface of the convolutions in some parts that it was impossible to remove them without tearing off the superficial layer of the cortex, while from other parts they were comparatively easily removed without any such damage to the underlying brain. There was a good deal of compensatory œdema of the lepto-meninges. The convolutions of both cerebral hemispheres were considerably atrophied, this was, however, not more pronounced in the frontal lobes than elsewhere; but possibly the shrinkage at the upper part of the central convolutions, more especially the ascending parietal, was a little more marked than in any other part. On palpation the cerebral substance gave rise to a greater sense of resistance than natural, and it was found to be unduly firm when sections were made into the hemisphere. On the whole, the grey and white matter appeared to be reduced in equal proportion, though in parts the atrophy of the former seemed the more pronounced.

No macroscopic changes were detected in the centrum ovale, basal ganglia or internal capsule on either side. The lateral ventricles were somewhat large in comparison to the size of the brain, but they contained clear fluid, and the ependyma showed no granular or other macroscopic change.

Beyond thickening of the pia-arachnoid, chiefly over the upper surface of its middle lobe, the cerebellum presented no abnormal features, nor did examination of the pons and medulla reveal anything unusual. There was no obvious lesion of any of the cranial nerves. The lepto-meninges at the base were not specially thickened, and there was nothing in the appearances met with to suggest that there had at any time been an acute basal meningitis.

The spinal canal was natural, as were the meninges, but the spinal cord was unduly soft and bulged markedly on section —a condition evidently the result of *post-mortem* change. The thoracic and abdominal viscera were natural, and no appearances were met with about the body, or in connection with any of the organs, which could be regarded as evidence of syphilis.

Microscopic Examination.—The central nervous system was carefully preserved for subsequent microscopic examination, and portions were prepared and stained by various methods, including those of Nissl, Marchi and Pal; in addition to which stains, such as aniline blue-black, hæmatoxylin and eosin, were employed. Portions of cerebral cortex, from the frontal and central convolutions of each hemisphere, thus dealt with, revealed on microscopical examination considerable thickening of the leptomeninges, with marked nuclear proliferation. Large bands of thickened pia extend deeply into the substance of the cortex at intervals, while in other parts the pia is separated from the surface of the brain, and no such thickened bands connect the two. There is marked sclerosis of the cerebral substance, with nuclear proliferation, and the walls of the vessels, both of the pia and in the brain, are greatly thickened. Profound degenerative changes of the neurons of the cortex exist; the cells are seen in all stages of atrophy, some almost beyond recognition, and there is obviously a great scarcity of them. All of the cells present are poorly supplied with chromatic material, and some of them, instead of being shrunken and atrophied, are obviously swollen, and in a stage of more recent degeneration. Similarly, degenerative changes are obvious in the axons, whose continuations can be traced, in many instances, as scattered, recently degenerated fibres, as they course through the corona radiata. No obvious degenerative or sclerotic changes are seen in the cerebellum, such as those that have just been described in connection with the cerebrum.

The most pronounced change met with in the spinal cord is a sclerosis of the postero-internal columns, limited to the cervical region, symmetrical, and occupying chiefly about the dorsal two-thirds of these columns. A very narrow band of sclerosis, on each side of the postero-median septum, extends ventrally from the main area of sclerosis towards the posterior commissure, but otherwise the ventral part of the postero-internal columns are free from sclerosis, as are the postero-external columns. There is distinct but much slighter thickening of the neuroglia in the region of the transverse area of the cord occupied by the crossed pyramidal tracts, and, like the posterior column sclerosis, this change is limited to the cervical region of the cord. Apart from the affection of the posterior and lateral columns, no other areas of sclerosis exist in the cervical region, and the thoracic and lumbar regions appear to be quite free from sclerosis, even the parts affected in the cervical region escaping here. Throughout the whole length of the cord, as revealed by sections taken at different levels, a few scattered, recently degenerated fibres are seen chiefly in the posterior columns, and still fewer in the lateral columns. In the lumbar cord, however, there is a considerable increase in the number of these fibres in the posterior columns, where they occupy almost exclusively the postero-external tract. Beyond pigmentation, and staining black in sections treated by the Marchi method, the cells throughout the spinal cord appear to be intact.

Case II.—E. G., male, aged $15\frac{3}{12}$ years, was admitted to the National Hospital, Queen Square, on October 29, 1897, under the care of Dr. Beevor, complaining of trembling of the limbs.

Family History.—Patient's father, a mulatto from the West Indies, is alive and very alcoholic. His mother, a white woman, died at the age of 50 from phthisis. No relation, either on the father's or the mother's side, is known to have suffered from any nervous or mental disease. Patient is one of a family of six. The first child was born dead at full term, but "looked as if it had been dead for some time before birth." The second pregnancy resulted in twins, both born dead, prematurely, at the seventh month. Then a girl was born alive, but she soon developed "snuffles," and at the age of four years developed right hemiplegia and fits, both of which still persist. She is now 17 years of age, has a depressed nose, and there is a distinct history of her eyesight having been affected for some time when aged 8 years. Patient was the next child. He was born apparently healthy, and never had any symptoms of congenital syphilis. One other child has since been born, but it died of whooping-cough at 9 months.

Previous Health.—He has always been a strong, healthy child, exceptionally intelligent. At school he passed the seventh standard and carried off many prizes. He has never had any other illness whatever; no infantile convulsions. Three and a half years ago he was knocked down by a passing cart and sustained a cut on his head. He was not unconscious at the time, and the wound promptly healed.

About three years ago his memory gradually began to fail. This was noticed first when he was sent out messages. He used to return having forgotten what he had been sent for. He also became much less bright at his lessons and ceased attending school. He was also noticed to "tremble" in his face and limbs whenever he was spoken to. Since then he has also had numerous spontaneous "trembling" attacks, during which the mental confusion has been more marked. There is no history of convulsions or twitchings of any sort. His memory and intelligence have deteriorated somewhat rapidly of late.

Condition on Admission.—Patient is a black-haired little boy. His complexion is very sallow. His eyes are prominent, giving him a somewhat startled expression, and his lips are large and thick. His hard palate is broad and normally arched. The teeth are well shaped and planted somewhat widely apart in front. There are no signs of congenital syphilis observable in the eyes or elsewhere. There is a small scar about half an inch long over the left mastoid process, about three-quarters of an inch from its tip, and there is a small horizontal depression on the mastoid process at this level, the result of the accident above noted. He can name objects readily, he can read and write, but in answering questions he is slow and stupid. His articulation is somewhat slurring and indistinct, and during talking there is marked fibrillary tremor in the muscles about the lips. He also licks his lips very frequently when talking, especially before beginning a sentence. He has no delusions or hallucinations; he is good tempered; he never speaks unless he is spoken to; he reads comic papers, but is very slow in appreciating the jokes. When, however, he does see a joke at last, he laughs loudly. He does not seem to care for play or amusement. His memory is very poor; he forgets which is his bed in the ward and tries to get into other patients' beds instead of his own. He has difficulty in repeating sentences read out to him, and tends to repeat only the concluding words of the last sentence read to him. When given a paragraph to copy out of a book he omits a number of words and mis-spells others. His hand-writing is tremulous.

He can smell asafœtida and peppermint normally in both nostrils, and he can taste sugar, salt, and quinine normally on both sides. A watch is heard at two inches distance in both ears. The fields of vision are normal on rough test; the optic discs and fundi are normal.

The pupils are dilated unequally, the right being much greater than the left. Both pupils are irregularly polygonal in outline. Neither of them contract either on exposure to strong light or during convergence. The external ocular muscles are normal; there is no squint and no nystagmus.

The facial movements, both upper and lower, are equal on both sides, but there is marked fibrillary tremor on voluntary movements, especially in the muscles about the lips and eyes. The fibrillary tremor is most marked when he opens his mouth widely. The muscles of mastication and the soft palate are normal; the voice is very tremulous. There is a coarse antero-posterior tremor of the tongue when it is protruded, not a true fibrillary tremor.

There is no anæsthesia anywhere to lightest touches or pricks on the face, trunk, or limbs.

All movements of the limbs can be executed well. There is no muscular weakness or atrophy. At rest there is no tremor, but on the slightest voluntary movement, as on holding his hand out in front of him, or on touching the nose with either index finger, there is considerable fine tremor of the whole limb.

The diaphragm and intercostals are normal, as also are the spinal and abdominal muscles. Patient's gait is somewhat ataxic and reeling; his back is slightly arched, and he stamps his heels in walking. He sways slightly on turning round rapidly, but can stand quite steadily with his eyes shut and feet together.

The knee-jerks are practically absent. It is difficult to be sure of their existence owing to the rigidity with which patient holds his legs in any position in which they happen to be. Occasionally, however, when the limbs happen to be relaxed, a very faint flicker of the quadriceps can be seen on tapping the patellar tendon, but not sufficient to cause any movement of the knee. There is no ankle clonus. The supinator-jerks, on the other hand, are extremely brisk on both sides. There is no jaw-jerk. The plantar, cremasteric and abdominal reflexes are brisk. There is incontinence both of urine and of fæces, but this appears to be due to patient's mental dulness, and not to any sphincter trouble. There is no onanism.

The external genital organs are very small, almost infantile. There is no pubic hair. The testicles are extremely small; only the left testicle is in the scrotum, the right is felt in the inguinal canal.

The heart, lungs, and abdominal viscera present no abnormality. The urine is normal.

On *December* 18th he developed scarlet fever, and had to be sent to a fever hospital.

Case III.—A. S., male, aged $17\frac{6}{12}$ years, was admitted to the National Hospital, Queen Square, on February 9, 1898, under the care of Dr. Ferrier.

Family History.—Both parents are alive and said to be healthy. There is no history of nervous or mental disease in any relation. Patient is the eleventh of fifteen children, of whom seven, including the two oldest, were born prematurely and dead, except one which survived a month. Of the eight full-time children, one died at birth, one died within a week after birth; three are alive and have no history of symptoms of congenital syphilis, two are alive but have had signs of congenital syphilis (one of these, a sister aged 14 years, had interstitial keratitis when seen on February 13, 1898). Patient is the only other full-time child. He was born apparently healthy ("a beautiful child") and was always very strong. He has had no illnesses whatever except measles. He is naturally of rather an irritable temper, but was clever at his lessons, passing the seventh standard when 13 years old. He never had any injury or accident of any sort.

About three years ago, when 14 years of age, he gradually became stupid and forgetful. If sent out on a simple message he would get confused and would return without having executed it. Then his actions became eccentric. On one occasion he took two caps from a draper's shop and brought them home with him; he also brought home an egg which he had appropriated from another shop. Ten months ago he bought a new knife and chopped up his watch with it, spoiling both knife and watch in the process. On another occasion he took off his trousers and in this state climbed on to the roof of the house. Within the last year he has become dirty in his habits, passing fæces in his clothes. Five months ago he was observed to walk unsteadily, reeling about but never actually falling down. About two months ago he seemed unable to speak for about an hour and a half—he "kept trying to talk, but could not utter any words." His articulation during the last two or three months has been noticed to be somewhat mumbling. He has never had any delusions or hallucinations at any time, and is said to sleep well.

Condition on Admission.—Patient is a well-nourished, fair-haired lad, height 5 ft. 2½ in. His expression is somewhat fatuous and his intelligence distinctly below par. When asked how many pennies there are in a shilling he replies promptly, "Don't know and don't care!" On being asked a second time he replies "Twelve." He appears disinclined to answer simple questions or to perform any desired act. He refuses to attempt to write, exclaiming loudly, "*I* can't write, *you* write!" When asked to spell his own name he says "Artur." His voice is loud though rather tremulous, and his manner distinctly rude. His articulation is characteristic of general paralysis; it is slipshod, consonants being frequently elided. During speech marked fibrillary tremors are visible round the lips; his tongue is also very tremulous. The hard palate is well formed and not unduly arched.

He can smell asafœtida and peppermint in both nostrils. He tastes quinine promptly on both sides of his tongue, pronounces it bitter, and spits vigorously on the bed. Sugar he tastes, but calls it salt. His mental condition prevents accurate comparison of taste on the two sides. Hearing appears normal in both ears. The visual fields seem normal on rough test; the optic discs are normal.

The left eye tends habitually to be in a position of slight external strabismus, but he can voluntarily bring the eyes parallel, and can execute all external ocular movements well. There is no nystagmus. The pupils are unequal (the right measuring 4 mm., the left 3 mm.), not quite central. They do not contract during convergence, nor when strong light is focussed on them. The left palpebral fissure is slightly narrower than the right, but both upper eyelids can be voluntarily elevated to their full extent. The muscles of mastication appear normal. The facial movements, upper and lower, are executed equally on the two sides, but are accompanied by much fibrillary tremor on voluntary movement. The soft palate rises normally on phonation. The tongue is protruded straight, but exhibits coarse rapid antero-posterior tremor.

There is no anæsthesia anywhere to lightest touches or pricks on face, trunk, or limbs.

The grasp of both hands is rather languid, but whether this is due to want of will-power cannot be determined. He appears able to execute all movements of the upper extremities, but all movements are accompanied by a fine rhythmic tremor. There is no tremor of the limbs at rest. There is no rigidity and no muscular atrophy.

The abdominal muscles appear strong. He can raise himself in bed from the lying to the sitting posture without using his hands. The spinal muscles are normal. Respiration is costo-abdominal in type, and normal.

All the movements of the lower limbs are executed normally as he lies in bed, but with the same fine rhythmic tremor as was observed in the upper limbs. His gait is feeble and unsteady, like that of an old man ; the feet are planted rather widely apart. There is no spasticity and no ataxy. He can stand with eyes shut and feet together. The knee-jerks are present and equal, rather subnormal. There is no ankle-clonus. The supinator jerks are brisk and equal. There is no jaw-jerk. The plantar and abdominal reflexes are brisk and equal; the cremasteric reflexes are absent. He occasionally passes urine and fæces in bed without calling attention to the fact, apparently from mental dulness.

The spine and cranium are normal. There is no tenderness on percussion, and no spinal curvature. The heart, lungs, and abdomen present no abnormalities. The genital organs are small for his age. Pubic hair is scanty. There is no axillary hair. The urine has a specific gravity of 1012 ; it is acid in reaction, contains no sugar, but has a distinct trace of albumin.

No signs of congenital syphilis can be detected in teeth, eyes, or elsewhere.

Patient's constant noisiness and shouting day and night necessitated his being discharged a week after his admission. Sulphonal, bromide of potassium and paraldehyde all in turn failed to quiet him.

Diagnosis.—The three cases described above were all undoubtedly examples of general paralysis of the insane. The differential diagnosis, when the occurrence of the disease in young people was borne in mind, presented no difficulty. A few of the possible alternatives may, however, be mentioned. None of the cases resembled cretinism in any degree save in the dementia, whilst they could not be classed amongst cases of congenital amentia, since all of the boys are known to have been possessed, previous to their illness, of intelligence above the average. Onanism, it is true, was observed in the first of the cases, but there is no evidence that it caused the dementia ; moreover, the diagnosis of gross central disease in this case was confirmed at the autopsy. The condition of infantilism, a rather superior type of myxœdematous idiocy, recognised originally by Lasegne, and of which Brissaud* has recently described a case, differs markedly from the condition in our patients, inasmuch as all of them actually retrogressed after attaining a degree of mental development, and were not simply permanently arrested in their growth, mental and physical. In addition, they showed the clinical peculiarities of speech and articulation which are characteristic of general paralysis of the insane.

It may be useful here to give a short summary of the symptomatology of general paralysis of the insane occurring in childhood and adolescence, more especially as the condition is very inadequately described, even in the most recent text books of nervous and mental disease. The following conclusions are derived from a study of the records of such cases as I have been able to gain access to in contemporary literature. The best bibliography of the subject is in an excellent monograph by Alzheimer,[27] who gives a summary of nearly all of the cases recorded up to 1895, together with three of his own (forty-one cases in all). Since Alzheimer's paper, fresh cases have been recorded by other observers (*vide* Bibliographical List). In all I have been able to collect records of fifty cases, exclusive of my own three cases above described.

The age of onset varies from the ninth year upwards.

Out of 49 cases in which the age of onset has been recorded—

4	cases	commenced	between	9	and	10	years of age.
3	,,	,,	,,	10	,,	11	,,
4	,,	,,	,,	11	,,	12	,,
2	,,	,,	,,	12	,,	13	,,
9	,,	,,	,,	13	,,	14	,,
2	,,	,,	,,	14	,,	15	,,
13	,,	,,	,,	15	,,	16	,,
5	,,	,,	,,	17	,,	18	,,
1	,,	,,	,,	18	,,	19	,,
4	,,	,,	,,	19	,,	20	,,
2	,,	,,	,,	21	,,	22	,,

The commonest age of onset, however, is from the thirteenth to the sixteenth year, roughly speaking, about the age of puberty. After the sixteenth year is past, the number of fresh cases becomes relatively much smaller as we approach adult life, amounting to only 25 per cent. of all cases occurring before the age of twenty-one. It would seem, therefore, that the age of puberty is a specially critical period in children of syphilitic taint.

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* Brissaud, "Leçons sur les Maladies nerveuses," 1893.

Both sexes are affected, apparently with almost equal frequency (twenty-five males to twenty-eight females). Wiglesworth,[20] who recorded five cases, all of them in girls, was of the opinion that the female sex is specially liable to this form of brain disease. The comparison of a large number of cases shows, however, that his statement is only partially true, since both boys and girls appear equally liable. But this is in marked contrast to the overwhelming preponderance with which the male sex is attacked in later life.

The relation of the disease to syphilis has been well established, and in the great majority of cases a family history has been elicited pointing to inherited syphilis:—in thirty-six out of forty cases in which a family history was obtainable, making a proportion of 90 per cent. in which a hereditary taint was actually ascertained. These figures probably under-estimate the real frequency of syphilis in one or other of the parents of the patient. It is, however, noteworthy that objective evidences of congenital syphilis in the patient's teeth, eyes, bones and elsewhere are conspicuous by their rarity, the syphilitic poison in such patients seeming to expend itself chiefly on the central nervous system. Indeed, we sometimes find (as in Cases II. and III. of my own) that whilst the patient himself shows none of the ordinary stigmata of syphilis, his brothers and sisters may exhibit congenital syphilitic lesions of the most marked kind, although they escape the brain disease from which the patient suffers.

Acquired syphilis is a very exceptional antecedent, but it has been recorded in at least three cases. In two of these, recorded by Sommer[11] and Rey[6] respectively, the children were infected by a syphilitic wet-nurse, whilst in the third, recorded by Hüfler,[18] the patient, when a little girl of five years, was infected by a kiss on the lip. (The symptoms of general paralysis in this case did not appear until the age of twenty.)

In a small number of cases there is a history of some fall or head-injury having preceded the onset of the disease (as in Case II. of my own), but it is doubtful whether such traumatisms have any influence beyond, perhaps, precipitating the onset of the malady in a patient already congenitally syphilitic. In other cases, again, a fall on the head has probably been merely an incident in one of the fits which sometimes usher in the disease, and frequently occur during its course.

The *Symptoms* of the disease may be classified into mental and physical.

Mental symptoms:—These typically consist in simple, progressive dementia in a child whose intelligence previously had been normal, or sometimes even above the average. The child becomes gradually dull, forgetful and apathetic, sometimes stuporose, but may have occasional bursts of passion. Grandiose ideas are, as a rule, conspicuous by their absence, therein contrasting markedly with the classical form of the disease in adults. Mild delusions and hallucinations may exist, but never of the extravagant type met with in adults.

Physical signs:—The patient's general development usually becomes arrested. Growth, which had previously been like that of a healthy child, either ceases or advances only very slowly. The genital organs remain infantile in type. In female patients, if the disease has set in before puberty, the catamenia remain absent throughout the remainder of the patient's life; if the onset of the disease has been after puberty, menstruation becomes irregular and soon ceases entirely. The general bodily condition, then, is one of arrested growth in an organism which had previously been normal in its development.

Fits or "congestive attacks" are common, and may be the first symptom of the disease. They may be simple attacks of unconsciousness without convulsions, they may be attacks of general "trembling" without loss of consciousness, or attacks of speechlessness (as in Case III.), or they may be typical and severe epileptic fits (as in Case I.). Death frequently occurs in one of these congestive attacks, in uncomplicated cases.

Cranial Nerves.—The special senses—smell, taste and hearing—are usually unaffected. Optic atrophy with its accompanying impairment of vision is not uncommon. The pupils, as in the adult form of the disease, are often irregular in outline and unequal in size ; the light reflex is generally sluggish or absent and the pupils may fail to contract during convergence of the eyes. Fibrillary tremors of the face, lips, and tongue are usually present when the disease is well established and are pathognomonic, whilst the articulation becomes typically slurring and slovenly, and the handwriting resembles exactly that of the adult general paralytic. In a few cases there has been facial irritability (Chvostek's symptom) on percussion over the seventh cranial nerve.

Sensory Functions.—The patient can usually feel light touches everywhere normally. General analgesia has been noted in a number of cases, *e.g.*, those of Hoch[34] and of Lührmann.[29]

Motor Functions.—Weakness of one or more limbs is very common and may be monoplegic, hemiplegic or diplegic in distribution. Such motor weakness often follows a congestive attack (see Case I.) and is increased by each successive fit. Unsteadiness of the upper extremities on voluntary exertion is common and may resemble the "intention tremor" of disseminated sclerosis. The lower extremities are frequently ataxic, sometimes, however, they are stiff and spastic, and in the later stages of the disease, when the patient is completely bedridden, permanent contractures may ensue (Thomson and Dawson[30] and others).

Reflexes.—The knee-jerks may be absent, or exaggerated, or normal, and this apparently whether the limbs be ataxic or not. (Thus in Cases I. and II. the gait was ataxic in both, yet in the former case the knee-joints were lively whilst in the latter they were practically absent.) The superficial reflexes are usually normal and the sphincters are unaffected, although the latter may become uncontrolled through the progressive advance of mental dulness.

The duration of the disease has varied from six months (Lührmann[29]) to eight and-a half years (Turnbull[2]). Out of twenty-eight fatal cases ten lasted over five years and fifteen lasted over four years. Other cases were recorded as being still alive after four, five, and seven years of the disease. The average duration of the disease is, therefore, longer than in adults.

As the disease progresses the patient gradually becomes more and more enfeebled, the limbs tend to become fixed by contracture, incontinence of urine and fæces supervenes, bed-sores tend to form, and death may occur from exhaustion. More commonly, however, the fatal result is brought about by a congestive attack, and this even before the patient has become severely paralysed in the limbs.

Morbid Anatomy.—The morbid anatomy does not differ essentially from that of the adult variety of the disease. Changes have been found in the brain, spinal cord, and peripheral nerves.

In the brain chronic lepto-meningitis is the rule, with firm adhesions between the pia-arachnoid and the surface of the convolutions on the convexity of the cerebrum, usually most marked in the central and frontal regions of the cortex. The affected convolutions appear somewhat atrophied. Internal hæmorrhagic pachy-meningitis sometimes occurs, as in the adult form, with the occasional formation of a sub-dural false membrane. Dilatation of the ventricles is common, and frequently also granular ependymitis.

Microscopically there is well-marked sclerosis of the cortex, both peri-vascular and neuroglial. Fatty and pigmentary degeneration of the pyramidal nerve cells of the cortex of the affected convolutions is usually found. Similar changes, though less marked, may occur in the nervous and connective tissue elements of the cerebellar cortex and of the basal ganglia.

In the spinal cord various systemic degenerations have been described. These may occur in various columns of the cord; for example, in the posterior columns, in the crossed and direct pyramidal tracts and in the ascending antero-lateral tracts.

Degeneration has also been found in the peripheral nerves.

BIBLIOGRAPHY.

(1) T. S. Clouston. "A Case of General Paralysis at age of 16." *Journal of Mental Science*, October, 1877, p. 419.

(2) A. R. Turnbull. "Notes of a Case of General Paralysis at age of 12." *Journal of Mental Science*, October, 1881.

(3) E. Régis. "Note sur la Paralysie Générale Prematurée." *L'Encephale*, 1883.

(4) M. J. Nolan. "Syphilitic General Paralysis." *Journal of Mental Science*, April, 1883.

(5) J. Wiglesworth. "Case of General Paralysis in a Young Woman, commencing at age of 15." *Journal of Mental Science*, July, 1883.

(6) Rey. "Cas de Paralysie Générale Héréditaire." Vide *Neurolog. Centralblatt*, 1883.

(7) Leidesdorf. "Ueber Psychiche Störungen im Kindesalter." *Wien. Med. Wochenschrift*, 1884, No. 27.

(8) E. Régis. "Un cas de Paralysie Générale à l'âge de dix-sept ans." *L'Encéphale*, 1885, v. 578.

(9) Léon Vrain. "Contribution à l'étude de la Paralysie Générale à début précoce." (*Thesis*) Paris, 1887.

(10) A. Strümpell. "Progressive Paralyse mit Tabes bei einem 13 jährigen Mädchen." *Neurolog. Centralblatt*, 1888, 122.

(11) Sommer. "Zum Zusammenhang zwischen Paralyse und Syphilis." *Neurolog. Centralblatt*, 1888.

(12) G. H. Savage. "General Paralysis of the Insane, simulating Cerebral Tumour." *Journal of Mental Science*, 1888.

(13) Davidoff. "Progressive Paralyse im Jugendalter." *Zeitschrift f. Psychiatrie*, 1889.

(14) Bjeljakow. "Pachycéphalie et Démence paralytique précoce dans un cas de Syphilis Héréditaire." *Zeitsch. f. Psychiatrie*, 1890, vol. xlvii.

(15) T. S. Clouston (2 cases). "The Neuroses of Development." *Ed. Med. Journal*, 1891, p. 1101.

(16) Régis. "Syphilis et Paralysie Générale." *Arch. Cliniques de Bordeaux*, Aug., 1892.

(17) J. M. Charcot et A. Dutil. "Paralysie Générale Progressive à début très précoce." *Arch. de Neurolog.*, 1892, 145.

(18) Höfler. "Progressive Paralyse bei einem 21 jährigen Mädchen nach vorhergegangener Syphilitischer Infection." *Deutsch. Zeitsch. f. Nervenkrankheiten*, 1892.

(19) MAJOR. "General Paralysis in a Boy." *Brit. Med. Jour.*, 1892, ii.

(20) J. WIGLESWORTH (5 cases). "General Paralysis occurring about the Period of Puberty." *Journal of Mental Science*, 1893, 355.

(21) BRISTOWE. "A Case of General Paralysis of the Insane at the time of Puberty." *Brit. Med. Jour.*, 1893, ii.

(22) WESTPHAL. "Ein Fall von Progressiven Paralysie bei einem 15 jährigen Mädchen." *Charité Annalen*, Jahrgang 18, 1893.

(23) SACKI. "Symptomenbild der Dementia Paralytica im Pubertätsalter." *Neurolog. Centralblatt*, 1894.

(24) HANS GUDDEN (3 cases). "Zur Ætiologie und Symptomatologie der Progressiven Paralyse." *Arch. f. Psychiatrie*, 1894, 430.

(25) J. MIDDLEMASS (6 cases). "Developmental General Paralysis." *Journ. of Mental Science*, 1894.

(26) VON KRAFFT-EBING. "Die Progressive Allegemeine Paralyse." *Nothnagel's Pathologie und Therapie*, 1894.

(27) ALZHEIMER (3 cases). "Die Frühform der Allegemeinen Progressiven Paralyse." *Allegem. Zeitsch f. Psychiatrie*, 1896, p. 533.

(28) KARPLUS. "Fall von Progressiven Paralysie bei einem 16 jährigen Mädchen." *Wien. Klin. Wochensch.*, 1895, 883.

(29) LÜHRMANN. "Progressive Paralysie im Jugendlichen Alter." *Neurolog. Centralblatt*, 1895, 632.

(30) J. THOMSON AND W. R. DAWSON. "Case of General Paralysis of the Insane in a Child." *Lancet*, 1895, ii., 397.

(31) E. L. DUNN. "Case of General Paralysis in a Girl, aged 9¾ years." *Journal of Mental Science*, 1895, p. 482.

(32) INFELD. "Demonstrirt einen 17 jährigen Knaben mit Progressiven Paralyse." *Wien. Klin. Wochensch.*, 1895, p. 832.

(33) RÉGIS (2 cases). "Deux nouveaux cas de Paralysie Générale Juvénile avec Syphilis Héréditaire." *Mercred. Med.*, Paris, May 22, 1895, 241.

(34) A. HOCH (2 cases). "General Paralysis in two Sisters, commencing at 10 and 15 respectively." *Jour. Nerv. and Ment. Diseases*, 1897, 67.

THE CEREBRAL CAPILLARY CIRCULATION.

BY JAMES CAPPIE, M.D.

THE thesis I venture to submit in the present paper is :—That metabolic commotion in the brain tissue is absolutely necessary to maintain an active capillary circulation through the cerebral vessels.

To support this heresy, as I suppose it will be called, I shall make use of a few of the most elementary principles in physics, and assume the soundness of one or two postulates. "Action and reaction are equal and opposite." "Pressure applied to a fluid is communicated equally in all directions." "Pressure by a moving fluid is in proportion to volume and velocity." "Movement takes place in the direction of least resistance." These will be my instruments of research.

I shall assume that the atmospheric pressure cannot directly bear on the contents of the cranial cavity, but that indirectly it can act on them through the blood-vessels that enter or make their exit through the skull. That pressure, therefore, must be directed, not, as in most other parts of the body, on the external surface of the intra-cranial vessels, but on their *internal* surface.

I also assume the soundness of the Monro doctrine as to the conditions that influence the mass of blood in the cranial cavity :—

"The ends of the lateral sinuses perforate the cranium at some distance from the arteries, that the returning blood might as little as possible suffer interruption in its course toward the heart. For any alternate stop of it, especially when occasioned by the stroke of the corresponding artery, must have had a worse effect on the brain than on other organs—not merely on account of the delicacy of the brain and the thinness of the veins, but because being enclosed in a case of bone, the blood must be continually flowing out by the veins, that room may be given to the blood which is entering the arteries. For, as the substance of the brain, like that of the other solids of the body, is nearly incompressible, the quantity of blood within the head must be the same, or nearly the same, at all times, whether in health or disease, in life or after death, those cases only excepted in which water or other matter is effused or secreted from the blood vessels, for in these a quantity of blood equal in bulk to the effused matter will be pressed out of the cranium." [1]

According to Dr. Kellie, Monro was in the habit of illustrating his doctrine "by exhibiting a hollow ball filled with water, and desiring his pupils to remark that not a drop of fluid escaped when inverted with its aperture downwards."

Having simply formulated his doctrine, Monro did not deem it necessary to fortify his position by immolating a hecatomb of victims. He seemed to take for granted that anyone with a fair modicum of intelligence who would take the trouble to grasp his propositions would not be able to do otherwise than come to the same conclusions as himself. The facts were patent; the inference seemed inevitable.

By and bye, however, the experimentalist came, and with him confusion. According to Dr. (afterwards Sir George) Burrows, such "opinions" were "erroneous," and "these errors obstruct our advances in the pathology of a most important organ." He argued that "the principle of subsidence of fluids after death operates on the parts within the cranium as well as upon those situated within the thorax or abdomen," and that "experiments and physiological considerations lead us to the conclusion that the quantity of blood within the cranium is extremely variable at different times and under different circumstances." [2] In support of these "opinions" he made a series of experiments on rabbits.

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[1] "Observations on the Structure and Functions of the Nervous System," p. 5.

[2] "On Disorders of the Cerebral Circulation."

A few months ago, if anyone wished to defend the position of Monro, it might have been necessary to enter on a lengthened argument. Fortunately, that does not now appear to be required. After having dominated and misled the opinions of physiologists for over half a century, an eclipse has come over the views of Burrows. Another experimentalist has come into the field, and after a series of elaborately conducted experiments, he has come to the conclusion that " in the living animal the Monro-Kellie doctrine is true."[1] A victory has thus been scored for common sense. At the same time it must rather be said that Hill's experiments have " completely overthrown " the views of Burrows, than that they have established the soundness of the Monro doctrine. The latter will ever stand securely on its own independent evidence.

In an essay like the present, I cannot enter fully into a historical detail of arguments for and against the opinion that the heart is not simply the dominant, but the sole efficient agent in maintaining the movement of blood through the vessels. There can be no doubt that at present the prevailing opinion is, " that the heart's action is continued onwards through the capillaries, and is sufficient to return the blood through the veins back as far as the heart."[2] It appears also to have become an article of physiological faith that in controlling local distribution, the vasomotor system of nerves is the all-efficient agent.

On the other hand, Professor Alison argued that " there must be some vital power concerned in *aiding*, not *opposing* the impulse from the heart . . . Local determinations are to be ascribed to a mutual vital attraction or affinity between the blood and the tissues of the body."[3] If anyone now ventures to defend the latter view, some expression of sarcasm will be the most likely comment. Nevertheless, not a little may be said in its favour.

On more than one occasion I have ventured to point out that the physics of the portal circulation may give important evidence in support of the Alisonian theory. My argument has been that as the blood from the portal vein has to traverse an extensive and minute plexus of capillary vessels before it can reach the hepatic vein, and so be sent on to the heart, considerable force will be required; that, in accordance with the law of pressure in fluids, that force must react as backward pressure on the blood in the mesenteric veins; that the walls of these vessels are not fitted to support such a strain; and that therefore, some supplementary agency that can act "in aiding, not opposing," the movement of the blood must exist in the liver itself. As opposed to this argument, I have not observed any answer that will bear a moment's consideration.

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[1] "The Physiology and Pathology of the Cerebral Circulation," by Leonard Hill, M.B., p. 35.

[2] "Cyclopædia of Anatomy and Physiology," vol. i., p. 671.

[3] "A Text-Book of Physiology," 5th edition, p. 219.

In the present essay, I have to submit further evidence in favour of the same doctrine from a consideration of certain points in the physics of the intracranial circulation. I have, however, a preliminary remark to make in regard to the mass of blood in the cerebral vessels. According to Professor Foster, the quantity must be "small, and making every allowance for rapidity of current, the interchange between the blood and the nervous elements must also be small." . . . In the rabbit, "while the weight of blood in the brain at any one time amounts to about 5 per cent. of the total weight of the organ, in the kidney it amounts to nearly 12 per cent., and in the liver to as much as nearly 30 per cent."[1] Now, this is surely a most misleading comparison. It is as if we were to take a mass of water flowing in a level marshy locality, and having wide banks, and comparing with it a fractional quantity of the same water flowing over a sloping bed and in confined banks, commend the former at the expense of the latter. Which of the two is endowed with the greater amount of kinetic energy? The one is of scarcely any mechanical value, the other can be used to drive powerful machinery. As between liver and brain the vital question is:—Which transmits the larger quantity of *arterial* blood through its vessels? No one will venture to assert that the hepatic artery can convey to the liver more than a fraction of the amount conveyed to the brain by the two carotid and the two vertebral arteries.

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[1] **"Text-Book of Physiology," p. 1134.**

For our present purpose we may take up the cerebral circulation after it has started from the circle of Willis. Without requiring to trace the course of individual vessels, we soon find the arterial blood flowing through the intricate meshes of the pia mater. On piercing the brain cortex the vessels are extremely minute, and their course is traced with difficulty. The blood, however, percolates the whole brain tissue, and we next find it flowing through the venous capillaries of the pia mater, and then through larger veins into the sinuses of the dura mater.

Several problems now present themselves for consideration. What forces maintain the flow of blood through the encephalic vessels? What are the conditions that influence the volume, velocity or direction of the blood currents, and thus determine the amount of intracranial pressure? How are the energising functions of the brain itself exercised? These problems are so intimately related that no one of them can be fully considered apart from the others, and it is almost a matter of indifference which we begin with.

In the first place, we may consider how the brain energises. If the sentiment "on earth there is nothing great but man; in man there is nothing great but mind" be true, then, in man's frame there is nothing great but brain. The activity of this organ is the condition of all conscious life. It affords the physical basis through which mental states and operations are revealed either to one's self or to the outward world. I have no intention, however, to make a survey of the whole circle of its potentialities. As bearing on the questions immediately before us, I shall restrict attention to one kind of activity —that associated with voluntary muscular movements; but if my argument here may stand the test of criticism, it will also apply to the centres of sensation and the field of association.

How, then, does a motor centre energise? The metaphysician may here be envied the facile way he overcomes—or ignores—all difficulties. The Will wills a movement and movement results! But although ready to allow the reign of metaphysics to be supreme in mind as *being*, the physicist claims sovereignty in everything that refers to the brain as *doing*. He regards that organ as he would any other working instrument, and may investigate the conditions that may influence the storage, intensity and liberation of its energy. He does not require to puzzle himself as to the nature of the nexus between volition and the energising motor structures.

I shall assume that every group of muscles, when in action, is correlated to a definite energising centre in the brain, and for the sake of clearness I shall take a single simple action—that of lifting a weight.

When a weight is lifted by the hand a certain strain is thrown on the contracting muscles. The strain is equal on the tendon that acts as a pulley-rope and on the fibres attached to the bone that plays the part of a lever. So far, the mechanical conditions are well understood. But if we go deeper we find that the dominant moving power resides in a limited area of the brain mass. It becomes a point of interest to learn how a soft pulpy piece of matter can energise so powerfully.

When a current of electricity is being tapped from the accumulator, it is assumed the molecular vibration or commotion in the energising medium must be inconceivably rapid. No molar motion can be detected in the dead-looking heavy material of the medium, but work, compared to which that done by the strongest muscles is the merest trifle, can thus be accomplished with ease and precision.

It cannot be otherwise with the brain. Its energy must be molecular, and the degree of commotion among its molecules will condition the amount of work done.

From the remarkable ease with which the functioning of the brain is carried on, we are apt to form a very inadequate conception as to the extent to which purely physical forces are at work through its mass. In ordinary circumstances we are no more conscious of their immediate working than of the weight of the quiescent atmosphere on the surface of the body. But when a blustering gale has to be faced we recognise the working of a potent energy; and when a gust of passion becomes a cyclone in the mental atmosphere, the existence of smouldering fires that may break out in volcanic explosion is revealed to us. In the exercise of muscular force we have special evidence of the powerful energy that may be dormant in brain cells, and we are driven to the conclusion that when a motor centre is in brisk action there must be intense commotion in its molecular structure.

But simple molecular activity can no more energise functionally than a string vibrating in a vacuum can produce sound. We must have metabolism as a whole in full play. This involves a sufficient supply of oxygenated blood, between which and the tissue there is an incessant interchange of material, and which requires every moment to be renewed.

The object of the circulation is here—as in every other part of the body—to assist in preserving the functional capacity of the organ by maintaining integrity of structure. It gives necessary stimulus in activity, and it affords material for recuperation after exertion. The supply of blood to individual organs has usually a direct relation to the requirement of the tissue, and unless proof to the contrary be afforded we may assume that the same principle holds good in the brain. Without deciding in the meantime how it is effected, I shall take for granted that the circulation will react sensitively to every change in the tissue. In sympathy with the degree of molecular commotion, it will be comparatively quiet or vigorously active.

If a man lifts a 56 lb. weight, and by sheer strength of muscle can whirl it round his head, and if the muscular force depends in the first instance on the energising of any part of the brain, evidently the strain within the latter must in some sense correspond to that in the muscles, in accordance with the law that action and reaction are equal and apposite. Molecular vibration must be at a high pitch, and the capillary circulation be correspondingly vigorous.

The movement of blood, of course, implies pressure, and from the extent of surface involved—from the myriads of vessels that are taking a part—the sum of the pressures must be very great. First in the large arterial vessels; then in the arterial capillaries of the pia mater; next in the capillaries of the brain tissue; lastly in the venous capillaries and larger vessels, we have movements in a multitude of directions, and therefore a considerable number of antagonistic pressures. Then, remembering that all these movements and pressures occur in a cavity whose capacity is rigidly limited, we have in addition the pressure of reaction. When the fluids are strongly flushing the brain and the vessels on its surface, the resisting cranial wall will throw the pressure backwards into the cavity. Such combined action and reaction constitute *stress*. This will be equalised through the whole cranial cavity on account of the fluid contents being continuous. It cannot be greater at one point than at another. The pressures may be legion; the stress is one.

The degree of intracranial stress, then, will depend on the volume and velocity of the moving fluids within the cavity. But we are told that not only is "the quantity of blood in the cerebral vessels small," but that "the interchange between the blood and the nervous elements must also be small." If this be the case we have here a serious exception to one of the fundamental principles of physics. It would prove that the brain is really an indolent organ, or that we may have powerful energy liberated without any correspondingly powerful physical agency.

The analogy between the phenomena of combustion and those of metabolism may be carried a long way. Some comparison is made in perhaps every text-book of physiology, but it usually refers to the chemical rather than to the physical phenomena. An important analogy, however, may also be traced in their physical aspects. The engineer in regulating his furnace, or the cook in managing her kitchen range, knows that it is not a large mass of air surrounding the burning fuel that determines the degree of heat. It is rather a restricted amount of air which is allowed to percolate the fuel in regulated *currents*. These are strong, and they are independent of all propelling agency. The air is sucked into and through the burning mass by an attraction greatly more powerful than the repelling force of the heat, and the rush may be strong enough to produce loud sonorous vibrations.

So with the cerebral circulation. Judging from the amount of blood that in a given time has to flow through the brain mass, we must infer that the rate of movement will be great. The quantity is to be determined, not by what can be squeezed out of the tissues after death, but by the number and size of the feeding vessels. Judged by this criterion it can be no "small" amount. According to the estimate of Haller, one fifth part of the whole mass of blood which leaves the heart is directed to the circle of Willis.

If any individual centre be small in bulk, its power will the more depend on intense molecular commotion, and if the capillaries are so minute that it is extremely difficult to trace their course, velocity of movement through them must make up for the limited volume of blood actually present.

The inference, then, seems inevitable, that when a motor centre is severely energising, its molecular structure must be in great agitation, and that the blood will be coursing through it with a strongly distending pressure.

But pressure, to be of any mechanical value, must usually be met by counter-pressure. We have seen how this is secured to the brain as a whole by the cranial wall, and the same principle will also apply to every individual centre. In the instance before us, where does counter-pressure or reaction come in? We have a moving power—the energising centre; we have a resistance to be overcome—the weight to be lifted; where is the fulcrum? If told it is to be found in the resistance afforded by the surrounding brain mass, we shall have to consider the conditions of stability there, but before discussing this point at present, let us look a little further.

When raising the pia mater from the brain surface, if a convolution be gently separated from its neighbour, a double plexus of vessels is seen to dip to the bottom of the sulcus. One set penetrates the cortex from without, the other makes its exit from within. On the surface of all the convolutions —in close contact with the dura mater—the same conditions are found. It is evident that unless provision is made to prevent such an accident, a block will be very apt to take place in the circulation of the pia mater, and therefore in the circulation of the whole brain. I am not aware that any such risk has received the consideration of physiologists. And yet, if the pressure force acting on the circulation be purely mechanical—if it bears on the blood only from behind, through the circle of Willis—the risk will be very serious. When the brain is active—with its circulation at the fullest, and its pressures at highest tension—any impulse that might cause further expansion of its mass to an almost infinitesimal extent would inevitably lead to occlusion of the pial vessels.

Recurring to the motor centre, so energising as to cause a heavy weight to be lifted, we found an outwardly distending pressure occasioned by the high velocity of moving fluid to be present through the structure. We also found that to enable its energy to be liberated with economy and precision, the centre must do its work under severe repression. We now find that the distending pressure must be such that it will not be allowed to interfere with the circulation through the pia mater.

If now we take a more comprehensive view of the active brain, the importance of its circulation in a purely physical aspect again obtrudes itself. During the waking state, when energy is simply potential but special functioning is in abeyance, each and every part of the brain must be ready, at less than a moment's warning, to respond to its appropriate stimulus, and to take on itself its special duty. This implies a full and steady circulation. Let the neurons expand and contract as they may, let their protoplasm and the dendrons or filaments vibrate ever so intensely, no response of consciousness will ever be given without the oxydising and stimulating blood. To maintain functional power, therefore, every structure must, as it were, keep a certain *hold* on the circulation. It will thus be enabled to resist efforts to deprive it of its legitimate share, and it will also refuse to allow the tissue itself to be readily pushed aside. In this way every part of the brain may stand in the relation of fulcrum to any local centre that may be actively working. If the hold on the circulation be relaxed, as will be the case during sleep, or after exhaustion, the stress through the whole cranial cavity will be lessened, and the working capacity of the brain itself be modified. Can all this happen if the blood circulates through the brain only under impulse from the heart? To say that it is extremely improbable seems to me to be a very mild way to express the negative.

The brain can be adequately studied only from a monistic point of view. Its functions and special centres are very numerous, but if the metaphysician insists that the unity of the mind must be regarded as an axiom, the brain in its successful working will be a veritable unit. Co-ordination, sympathy, harmony, must reign through every part. Between molecular activity and the capillary circulation, correlation will be absolute. They constitute one agency. The former may be the dominant factor, but if the exercise of function is to be full and exact, the response of the latter must be direct. As there is mutual interchange of material, so there is mutual attraction—mutual control—mutual dependence.

It is no more necessary for one to unravel all the subtleties of this mutual relation in order to prove its reality, than it is necessary for the physicist to explain all the secret transformations of energy that take place in his machine. Suffice it to say, that in the movement of sap in the leaf of a plant we have evidence that fluid and molar motion can be directly caused by molecular activity, and we have only to suppose that (for example) in the tissues of the brain the changes are immensely more rapid and powerful to recognise a legitimate analogy. When metabolism is in full play "the physical interchanges between the fluids within and outside the capillary walls, the chemical affinities, the attractions and the repulsions, the inconceivably rapid oscillations of the molecules, all go to constitute a force which draws the blood onwards and *actively* transmits it through the capillaries."[1]

Other reasons may be given why such a relation should be considered not simply likely but necessary. Obviously, the *ratio* of the heart's action must be the same to the whole interior of the cranial cavity. Its greater or less force or frequency cannot influence the determination of the currents through particular vessels. It may maintain a moving power through the larger arteries, but it cannot affect the distribution through the brain mass.

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[1] *The Monist*, April, 1897.

Again, and possibly this argument may by some be considered the most important of all, there appears to be an absence of vaso-motor control over the brain circulation. According to recent researches the tyranny of these nerves has been more than questioned. Their very existence in the most important region of the body is denied. "Baylis and I," says Mr. Leonard Hill, "have been entirely unable to find any evidence of a vaso-motor supply to the brain . . . . In a recent and exhaustive research, Gulland has failed to demonstrate by any known histological means the existence of any vaso-motor nerves in the pial vessels."[1] If such views shall be confirmed by other observers, they should afford a cogent argument in favour of Alisonian principles. If vaso-motor pilotage be dispensed with, what can possibly determine the direction of the circulating blood? Does it meander at its own sweet will, or is law still dominant, and must it move in the direction of least resistance? Does it simply *soak* into the tissues and then ooze out again? Do motor, sensory and other centres receive each a certain uniform quantity, apart altogether from the amount of work it performs, and, therefore, whether the need be great or small? To think so is to suppose that all physical law in the cranial cavity is suspended, and that metaphysic is as dominant in the brain itself as it is in the mind. The laws that affect moving fluids must here hold sway as absolutely as on the mountain top, or in the depths of the sea.

This leads us to point out an interesting and most important service afforded by the pressure of the atmosphere. One of the obvious purposes to which this agency is put is to counteract the unfavourable influence of gravitation on the circulation of the brain when the body is erect. In the words of Dr. Kellie, it is " a force which must be constantly operating to maintain the plentitude of the vascular system within the head." As I have already mentioned, it bears on the internal surface of the vessels. It therefore acts on the arterial blood in the direction of the circulation, and it is opposed to that in the veins. So far as determination to and from the head is concerned, the pressure on one set of vessels is balanced by that on the other. Thus the pressure through the venous sinuses will not interrupt entry by the arteries. So long as there is no disturbing influence within the cavity, the balance will also be maintained through all the encephalic vessels. But if any influence at the capillaries may give rise to an *attractive force*, then, in relation to the atmospheric pressure, it will become *suction*, and unless physical obstacles interfere, the blood will be compelled to move toward the focus of attraction. The effect in this respect will be as certain as it would be on the fluids in the neighbourhood of an exhausted cupping-glass on the surface of the body. Toward any energising centre, therefore, the atmospheric pressure becomes an important adjunct to the action of the heart, and the direction of least resistance for the blood to move in will be *in the line of greatest attraction.*

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[1] *Op. cit.*, p. 45.

The trend of physiology is so completely in the direction of *experimental* research, that an apology might almost be expected from me for presuming to submit what may be considered important issues without any attempt to verify my conclusions in the laboratory. " Such a subject as the hydrodynamics of the cranium," says Leonard Hill, " can only be investigated by experiment." I answer, as dogmatically, that by simply making use of those elementary physical principles that relate to moving fluids, and applying them to the peculiar conditions we find within the skull, more direct light will be thrown on cerebral physiology, and especially on the correlations of mind and brain, than will ever be made by the most careful experimenting.

Were scores of experiments made on animals after their death, which might seem to prove that by a force not greater than that of the heart's action, some bland fluid may be made to flow from the carotid artery into the jugular vein, my faith in the Alisonian doctrine would not in the least be disturbed. It will be simply impossible to arrange conditions at all resembling those present in the living brain. The absence of strain, and of all the antagonistic pressures, would thoroughly vitiate any conclusions that might be drawn from such facts.

In regard to Mr. Hill's own experiments for the purpose of measuring intracranial pressures, they, too, are seriously vitiated by the simple fact that the animals operated on have previously been made unconscious. If unconsciousness exists, the molecular energies of the brain must be immensely reduced, and therefore the conditions that can influence active circulation will be in comparative abeyance. The principal, if not the sole source of intracranial pressure is the moving blood, and if the intimate relation of the capillary circulation to molecular activity be ignored, the results arrived at cannot be otherwise than partial and inconclusive. We get "the play of Hamlet, with the Prince of Denmark left out." The experiments have evidently been made with painstaking care, but from their very nature—from the impossibility of including the most essential factors—they will fail to give data more likely to throw light on the functioning brain than can be got without them.

FOUR CASES OF FRIEDREICH'S ATAXIA WITH A CRITICAL DIGEST OF RECENT LITERATURE ON THE SUBJECT.*

BY J. MACKIE WHYTE, M.A., M.D.
Physician, Dundee Royal Infirmary.

SUMMARY :—*A group of three cases of Friedreich's ataxia; an isolated case; tabular comparison; remarks on some of the symptoms, ataxia of voluntary movement, static ataxia, Romberg's sign, nystagmus, choreiform jerks, athetoid movements, deformities of upper and lower extremities, and of spine, condition of sensory system, mental state, reflexes, order in which the symptoms appear; their mode of advance; period of onset; increasing complexity of diagnosis; diagnosis of isolated cases; diagnosis from chorea, from hereditary cerebellar ataxia; diagnosis as affected by indications of mental defect; diagnosis from disseminated sclerosis, from locomotor ataxia, especially in children, from cerebellar disease; pathological anatomy; theoretical views as to pathology of the disease; treatment; literature.*

THE list of family diseases of the nervous system is now fairly lengthy. Higier,[78] one of the most recent writers on the subject, makes out a list of thirteen groups, as containing the more important representatives, and some of these consist of several sub-groups. It is common to find, as Marie puts it, that each family even of fairly well-defined type such as Friedreich's disease has its own special traits; consequently the grouping of affected families is more or less arbitrary, and in many cases, as yet premature. As we shall see further on, the records of transition forms, connecting one class of disease with another, are multiplying. What is wanted is the continued careful recording of clinical cases of those rarer diseases with, if possible, exhaustive autopsies.

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Friedreich's ataxia is a well-marked clinical type; many cases belonging to it have a "family" character, many are "isolated"; only a few are, properly speaking, "hereditary." This paper first gives a clinical account of four cases of Friedreich's ataxia; then follows a discussion of a number of the more interesting points in connexion with the symptoms, diagnosis and pathology, along with a critical review of the recent literature.

A. A GROUP OF THREE CASES OF FRIEDREICH'S ATAXIA IN ONE FAMILY.

The parents of the first three cases of Friedreich's ataxia about to be described were Irish. They lived in Ireland till after the birth of their fourth child. The father, a factory worker, was a heavy drinker, and died at 50 of liver disease. The mother, likewise alcoholic, died of pneumonia. She suffered also from gastric ulcer, obstinate pruritus, and eczema of face and neck. The eldest son informs me that his father used to say he knew of no nervous affection previously in their family. No history of insanity, fits or any other nervous disease can be obtained. The circumstances seem generally to have been very poor, such as are common to thousands of Irish in Dundee and Lochee. The patients belong to a family of nine, accounted for as follows:—

(1) First child, premature, died at two months.

(2) HUGH—*ataxic*.

(3) MARY—*ataxic*.

(4) Nora, married, died, aged 25, in childbirth; showed no ataxic symptoms; had three children, eldest alive and healthy, the other two died when a few days old.

(5) Maggie, married, subject to headache since her marriage; two premature children, dead; one alive and healthy.

(6) John, died aged 13 of typhoid; had shown no ataxic symptoms.

(7) Agnes, healthy, recently married.

(8) James, healthy (aged 17).

(9) WINIFRED, *ataxic*.

Numbers 5 and 8 show nystagmoid jerkings; their knee-jerks are normal. All were suckled by the mother. Mary shared her mother's milk with another child.

Case I.—Winifred M'A., aged 15, a mill-worker.

Patient's History.—She seems to have had good health until March, 1888, when she was sent into the Dundee Infirmary as suffering from typhoid fever. A brother, aged 13, had died at home of this disease. Her illness had begun with rigors and slight diarrhœa, and she had lain at home for three weeks before admission to the Infirmary. She was there found to be underfed, and to be suffering from slight diarrhœa, but beyond this there were no definite signs of illness, and she was soon discharged much improved.

A short time after this her father and eldest brother observed the first symptom of her present illness, namely, some difficulty in walking. Her mother also spoke of the way the girl kicked when asleep. Very gradually a change in her speech developed, so slight that it would probably have escaped notice, had not her brother Hugh been on the look-out for symptoms of the disease from which he and his sister Mary had long suffered. On the sensory side there were no complaints with the exception of some pains in the back of the thighs, occurring in the spring of 1894.

In July of that year I sent her into one of my wards in the Dundee Infirmary, and since then she has been more or less constantly under my observation. During the past two years the girl has frequently stumbled and fallen when running, and this seems to have been always in imperfect light, in the early morning or in the evening. Moreover, she says the staggering is worse when going to her work in the morning, both in summer and winter, rest having apparently a greater influence than fatigue in increasing it.

Present condition (February 21, 1897). Fairly well grown; expression of upper part of face bright and intelligent. The lower jaw usually droops and the mouth is partly open. Cheeks well coloured; nutrition rather below the average. (This is easily explained by her home conditions.) Bones seem well formed; muscular development rather feeble, but not specially so in any locality.

Nervous system.—The following conditions are to be noted on the *motor* side. When *sitting*, slight swaying movements of the upper part of the body occur; the hands can lie perfectly still. When *standing*, patient prefers to keep the feet some distance apart, and can then balance herself steadily enough if her eyes are open. She fixes them on some object in front of her. There is swaying of the trunk and head, made much worse when the feet are brought close together. If the eyes are then shut, patient at once falls over to one or other side, or forwards. The tendons on the dorsum of the foot are seen to contract (*danse des tendons*), and the toes grasp the floor; the great toe, however, is often over-extended. Balancing on one leg is quite impossible. Her head is always bent forward a little, whether sitting, standing, or walking. Her *gait* is distinctly awkward and ungainly. The steps are of varying length, her feet are sometimes wide apart, sometimes they knock against each other; she cannot keep to a straight line. Her feet are brought to the floor with a slight stamp, through a jerky movement with the character, not of spasm, but of inco-ordination. The staggering character is more marked when walking backwards, and still better if the eyes are closed. She can turn round fairly well, and can stop quickly when told to do so.

All *voluntary movements* can be carried out in a way, but the *ataxia* which is so marked in walking is likewise manifest in other directions. For instance, when patient is told to pick up a small thread off a smooth wooden surface the hand and fingers oscillate a little, and she prefers to rest them on the surface before catching the object; there is an awkwardness about the act. When told not to touch the board, the hovering movement described by Charcot is shown. Apart from this ataxia, however, she can carry out her intentions—can button her clothes behind her back, thread a needle, etc. A full cup is slightly spilt when she carries it, but the oscillations are not increased when she puts it to her mouth. When put through other tests for inco-ordination in the upper extremities with the eyes shut, there is a decided defect. When told to touch the tip of her nose, for instance, her forefinger first reached the upper lip at its lower border. My notes show a distinct increase in the ataxia of the upper extremities since 1894. When lying down she can touch objects with her heel or toes without very marked inco-ordination.

Involuntary movements include some already mentioned—the swaying of the body, the oscillations in carrying out a voluntary movement. But there are also occasional choreiform twitchings in the face, especially drawing up the right side of the mouth, and some jerky movements of the same nature have been observed in the head and fingers. To this class, too, belong the evidently very marked jerkings that occur during sleep. Patient is quite unaware of them, but they must interfere considerably with the repose of her bed companions. They have been continuous since the onset of the illness.

Certain involuntary movements are seen in the eyes when they are strained by being directed to the extreme right or left, a slow nystagmus, about one oscillation per second. When the eyes are fixed on an object above or below the horizontal line, a very fine oscillatory movement can be detected.

When the tongue is protruded, constant slight movements are seen.

Disturbance of speech is to my ear decidedly more marked than it was in 1894. And yet it is not very bad, especially when patient is paying attention to it. Her voice is deep-toned, the words are uttered slowly, there is rarely any slurring; long words or combinations as "post office savings bank," "articulation," can be uttered with a single effort. A stranger might find little difference between her speech and that of other girls of her class, but my own knowledge and her friends' statements show some deterioration to have taken place. She can write legibly, but the writing is tremulous and uneducated; she holds the pen or pencil in a clumsy way, but one must remember that she has hardly written a line since leaving school three or four years ago. I do not think her writing has grown worse since I first knew her.

Sensory Functions.—Patient used to have a giddy feeling at times, but she is now free from that. There are no abnormal sensations whatever and the various forms of cutaneous sensibility are normal.

The muscular sense is affected; patient is a good deal astray in trying to localise the position of arm or leg when blindfolded. (In 1894 the sense of posture in the upper extremities was fairly correct.) She constantly makes mistakes in trying to distinguish between different weights with her legs, but with her arms is fairly correct. Hearing, sight, and taste are normal; deglutition, defæcation and micturition are normal. Patient has not yet menstruated.

The *knee jerks* are completely absent and have been since I saw her first. Superficial reflexes normal.

Condition of Muscles, and Deformities.—There is no sign of flabbiness and no muscular atrophy can be detected. But there is an increasing deformity about the hands. When the right hand is extended, there is a very slight flexion of the wrist, a slight hyper-extension at the metacarpo-phalangeal joints of the fingers, especially the fourth and fifth, and a gentle flexion of the inter-phalangeal joints. The little finger shows a decided bend forwards. The fingers can be well separated from one another, and the wrist moves freely in all directions when the fist is closed, but by no effort can patient extend the phalanges completely. The thumb is not affected in any way. The same description applies to the left hand, though here the deformities are rather less marked.

This condition of the hands is very distinctly worse since 1894.

The feet show a very high instep and prominent dorsum when patient is resting, not so marked when standing. There is no talipes and no hyper-extension of the great toe beyond the occasional involuntary movement before mentioned.

A slight spinal curvature is present, convex to the left in the middle dorsal region, convex to the right in the upper dorsal and cervical regions. The bend forwards of the head is due to a slight kyphosis.

Grasp is good on both sides, and the muscular power of the legs seems to be normal. Patient is easily tired, but works for the full day, earning seven shillings a week as a shifter.

Electrical reactions are normal, both with faradic and galvanic currents.

Her psychical condition is normal; intelligence rather above the average.

Case II.—Hugh M'A., aged 33, a mill-worker, began to walk when 18 months old. Between his third and sixth year he suffered from some illness "like water in the head," and was for two years in bed. He went to school when 6, and remained there till he was 12, learning with normal facility.

At the age of 9 he had a bad fall on the back of his head, from a cart, and was rendered nearly unconscious, but there were no bad after-effects. On leaving school he began to work in the mill, and showed some nervous symptoms. Other workers were afraid he would fall among the machinery, and some suspected him of smoking. There do not seem to have been any ataxic symptoms sufficient to attract attention before this.

He soon got into the way of holding his head forwards, with his eyes bent on the ground that he might balance himself better; he preferred to keep his hands stretched out, the palms being directed forwards when walking.

From the age of 14 he found some difficulty in picking things up. There was not much change in his condition for the next four years, but he found that mill overseers were rather shy of engaging him in case of accidents. Between 17 and 19 he thinks he improved, but after that be became markedly worse, and from the age of 20 walking has been impossible. About that time he had loss of sensation in his feet, and got a burn on one of his soles through standing on a hot coal. For a long time he has suffered from numbness and feelings of creeping and prickling in his feet and legs, not in the arms. Often about two hours after going to bed he has a sensation of blood rushing up to his head, and his heart seems to stop. His legs sometimes start involuntarily at night. He has had no lightning pains, no girdle pains, no vomiting, no disturbance of bowels, no affection of vision. From about the time at which he lost the power of walking he has had some difficulty in micturition.

On August 1, 1884, he was admitted to the Dundee Royal Infirmary, and though the notes then taken are brief and evidently imperfect, it is worth while reproducing them for comparison with his present state :—"Head hydrocephalic in shape, 22 inches in circumference. Dorsal spine shows right lateral scoliosis of very exaggerated character ; sternum raised ; left lower ribs touch crest of ilium.

"*Sensory Functions.*—Tendency to coldness in feet ; formication in soles ; tingling sensations in legs and feet ; anæsthesia to touch and pain below level of iliac crests ; pupils large, sight good, hearing good.

"*Motor Functions.*—Drop wrist well marked on right side when hand held out, muscles of fore-arm and ball of thumb flabby, diminution of grasping power, no power in lower limbs except slight power of extension of hips and knees.

"*Visceral Reflexes.*—Normal except that patient sometimes takes half-an-hour before he can micturate. No skin or tendon reflexes in lower limbs.

Slight tendency to waste in muscles of legs. Sweats a good deal."

In July, 1894, I examined him minutely, and on comparing the notes taken then with those made now, I find very little alteration.

State on Examination, February, 1897 (see *figs.* 1, 2 *and* 3).—An under-sized, fairly nourished man, face somewhat flushed, expression intelligent, features thick, this being especially seen in lips, head large, brachy-cephalic, 59·4 cm. in greatest horizontal circumference, held well forwards. His usual *attitude* is sitting in an arm-chair, with his arms resting on a table in front of him. If he has no table before him he likes to twist his arms round the arms of the chair, to keep himself from sliding off. When thus supported in a sitting position his left shoulder is depressed, and if the *spine* is looked at (see fig. 2) it is

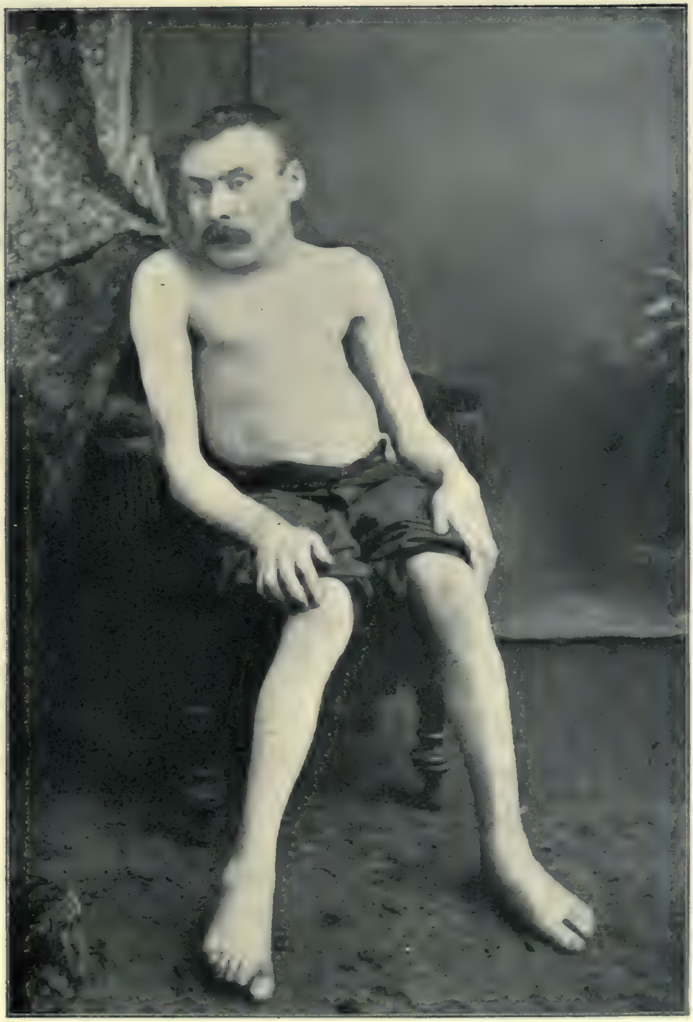

Fig. 1.

Case II.—General helplessness indicated. Patient is supported by man behind curtain.

seen to be curved (convex aspect) to the right, between the first and twelfth dorsal vertebræ. A slight compensatory curve the other way is seen in the lumbar region. The spine also shows a marked convexity backwards, in the upper dorsal regions, and a concavity (lordosis) in the dorso-lumbar. The upper four dorsal and the lower cervical spines are nearly horizontal.

When lying as straight as possible on his back the lower end of the sternum is very prominent, 7 cm. higher than the upper end. The sternum is straight, except the xiphoid, which is bent back. Intercostal spaces are narrow. No prominences at costo-cartilaginous unions. The hand can be easily passed behind the back on account of the lordosis. The ribs on the left are depressed to the iliac level.

Extremities.—(*a*) Upper. Muscles moderately well developed and pretty firm. No marked atrophy, though the interossei seem thinner and softer than any other set. No contractures, no fibrillary twitchings; no rigidity; passive movements possible in any direction.

(*b*) Lower. Muscles fairly firm, but considerably wasted, apparently from disuse. Right calf 25·6 cm., left 27 cm. in circumference; legs lie helplessly in bed, generally flexed at knee; foot shows a permanent deformity, the shape closely resembling that of *pes cavus*, arch extremely well marked on both sides, less on right than left; dorsi-flexion of first phalanges of toes, clawing of other phalanges to some extent. The deformity is less marked when the feet rest on the floor. On first exposing the right foot, a fibrillary tremor was noticed in the muscle on the inner border close to the arch (short flexor of great toe). Limbs very cold, and red or blue below the knees.

Motor Functions.—When in bed the patient prefers to lie on his right side, curled up. Cannot lie on left side longer than half an hour at a time. Has the greatest difficulty in raising himself, or turning round. In trying this he makes enormous efforts, painful to witness, and becomes very much flushed. The voluntary movements of the trunk are thus very jerky; he tosses himself round from one position to another.

Respiratory movements require a distinct effort; when sitting quietly with his table before him, both inspiration and expiration are audible through the room, a distinct pause separating them. The breathing is nasal; with each inspiration the head is jerked up a little. He is said to breathe more heavily and to snore at night. Power in the muscles of the neck seems well preserved; head can be moved in any direction. Occasionally there is a slight choreiform vibration of the head, especially on making a voluntary movement. Swaying movements of the head and body are constant when sitting. No choreic twitchings of face, or other disturbance of facial movements.

Voluntary movements in the *arms* moderately active, but not so powerful as one would expect from the appearance of the muscles. Grasping power much diminished; free movement at shoulders, elbows and wrist. When told to straighten the fingers, the hand resembles the *main en griffe* of progressive muscular atrophy (see fig. 3). There is flexion at the wrist, over extension at the metacarpo-phalangeal joints, and flexion at the first interphalangeal joints. There is dorsi-flexion of the second phalanx of the thumb, and also of the ungual phalanx of the right mid-finger. (This the result of an accident.) The index is less affected than the other fingers, and the condition is more exaggerated on the right side. In extension the fingers are kept slightly apart, but they cannot voluntarily be completely adducted or abducted. The wrist and fingers can be quite easily straightened by an external force. The extensor tendons can be traced up the lower third of the fore-arm, and on the front the tendons of the palmaris longus and other flexors stand out. The palm is hollowed.

Movements of fingers and arms, in trying to pick up or touch objects, are badly co-ordinated. Patient likes to have the arms supported on the table, etc.; when this is done there is not much oscillation. Sense of posture in upper limbs defective.

As for the *lower limbs*, walking is quite impossible. Slight power of flexion and extension at hip. None at knee or ankle, very little at toes.

Sensory Functions.—In the legs there is slight diminution of acuteness in sensibility to touch, but pain is felt normally. No lightning pains; some slight paræsthesia in lower limbs is noted in the history.

Reflexes.—No knee or Achilles jerks, plantar reflex well marked on right, slight on left. Cremasteric and abdominal reflexes marked. Visceral reflexes mormal apart from delay in starting micturition.

Sight.—Normal; pupils act readily to light and accommodation; a moderate degree of nystagmus, consisting of irregular jerkings on fixing eyes on an object, to the side.

Hearing.—Slightly dull (this also the case in 1894). Hears watch at 12 in. on right side, at 3 in. on left. Occasional sound of rushing water in his head.

Tongue.—Flabby, marked by teeth, slightly furred on dorsum; when protruded a constant fine tremor pervades its whole extent.

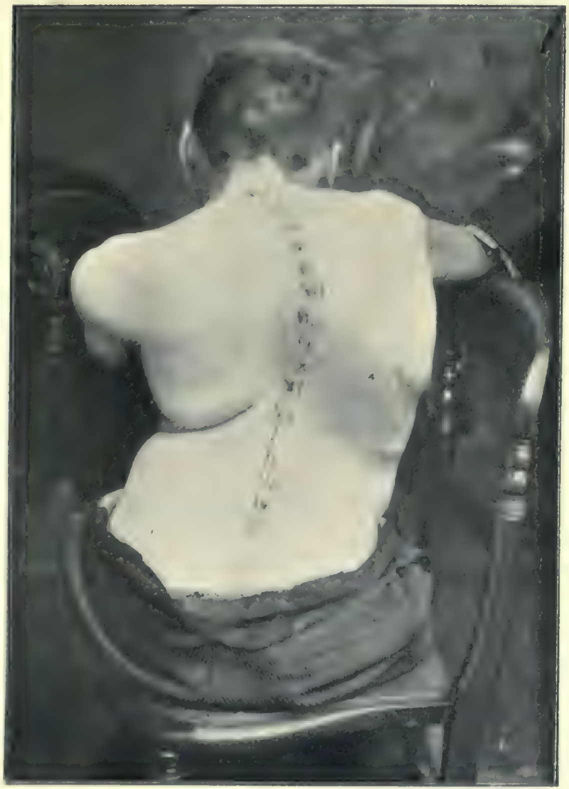

Fig. 2.

Case II.—To show Scoliosis and Kyphosis.

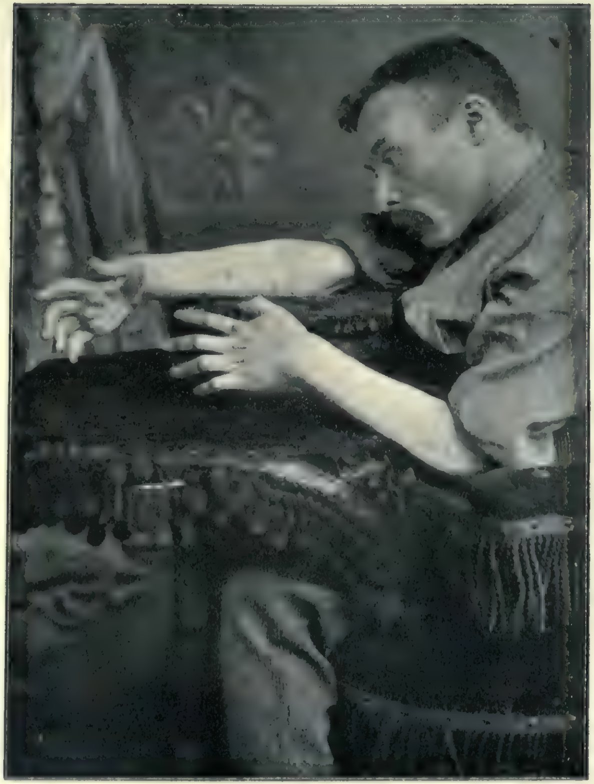

Fig. 3.

Case II.—To show *main en griffe.*

Speech is markedly disturbed. One requires to attend closely to make out all he says, though if his attention is directed to words, he speaks much more clearly. The voice is monotonous, the words are somewhat slurred, not scanned. "Remember" becomes "rememr," "Whether" becomes "wher." "British constitution" becomes "Brish constution." "A dainty dish" becomes "a den dish."

Psychical state is normal. Patient is said to be rather irritable, and to make what the others consider undue demands from his being the eldest of the family. His intelligence is quite unaffected.

Electrical reactions with galvanic current. Extensors of left forearm KCC 7, ACC 12, KOC 8, AOC 9. Flexors of left forearm, KCC 9, ACC 9, KOC 12, AOC 13.

Heart—Sounds normal; position altered, apex being in third left space external to nipple.

Case III.—Mary M'A., aged 31, formerly millworker (figs. 4 and 5). *History:* Patient had inflammation of the bowels, measles and whooping cough in infancy, but there was nothing indicating any ataxia or paralysis until she was fifteen. At this age, in the month of July, she was taken ill one morning when on the way to work, feeling cold and suffering from bleeding at the nose and mouth. She became unconscious that night, and remained so for about six weeks. During the next eighteen months her mind was enfeebled, and she could not walk steadily; she managed, however, to do some work in the mill, and in course of time her mind recovered its former clearness. The staggering, however, increased to such an extent that at the age of twenty-one she was refused further employment. From the beginning of her illness she was subject to cramps and to jerkings in the legs at night.

At the age of nineteen, and again when she was twenty-one, she was a patient in the Dundee Royal Infirmary on account of spinal curvature.

Until she was twenty-six she was able to get about on her legs, pushing a chair in front of her, but during the last few years she has been unable to walk at all. She says that her hands have both been weak—the right more than the left—ever since the acute illness which marks the beginning of her disease. Still she could sew, although her hands were shaky; she has never been able to read or write, her school education having been very imperfect and short in duration. Since July, 1894, she had been almost constantly under my observation, in the Dundee Infirmary and the Parochial Hospital.

Present Condition.—A well-nourished, fresh-coloured woman, with cheerful expression, ruddy complexion and numerous freckles on face and neck. Her hair is light brown, strong and abundant; her nose is twisted from a fall four years ago, and on her right cheek is a scar, due to another accident. Through this scar there is a puckering of the cheek below the inner part of the right eye, when the lids are firmly closed; there is also a slight drawing-up of the right angle of the mouth.

The head is well-shaped, and measures 55·4 cm. in the greatest horizontal circumference; it is kept bent forwards through the spinal curvature, but patient says she was accustomed to hold it so because she was ashamed of her staggering.

Her usual attitude is sitting up in bed, with the back well supported by pillows; the spine shows a marked dorsal curvature, with convexity to the left, extending from the sixth dorsal vertebra to the second lumbar, the deviation at its maximum amounts to 1½ inches; the ribs project backwards to a marked degree on the left side, and are well separated from one another. On the right they are crushed together, and the twelfth rib is on a level with the iliac crest. In the upper dorsal region there is a slight curve with convexity to the right.

Some kyphosis is present in the upper dorsal spine; this can be removed partially, and for a very short time, by an effort of the patient.

The dorsal curvature is somewhat less pronounced when she is raised up by the arm-pits.

She complains a little of soreness, on pressure over the spine in scapular region.

The mammæ are well developed, and there is no striking asymmetry when the chest is examined from the front.

Extremities.—Upper extremities show, when at rest, no deformity; the bones are well shaped, the muscles seem smaller than normal on the back of the fore-arm. The lower limbs, on the other hand, show a deformity about the foot, talipes equino-varus being present on both sides. The legs below the knees are more cylindrical than usual, from a want of development of the calf; the antero-lateral group of muscles is deficient in bulk; the heel is drawn up, some transverse wrinkles of the skin being present over the lower end of the tendo-Achillis. The hollow of the pedal arch is very much greater than usual, and this is so even when patient is made to stand. Most of the grooves on the sole run from within, outwards and forwards. The great toe, especially the right, is always over-extended. It can be brought voluntarily

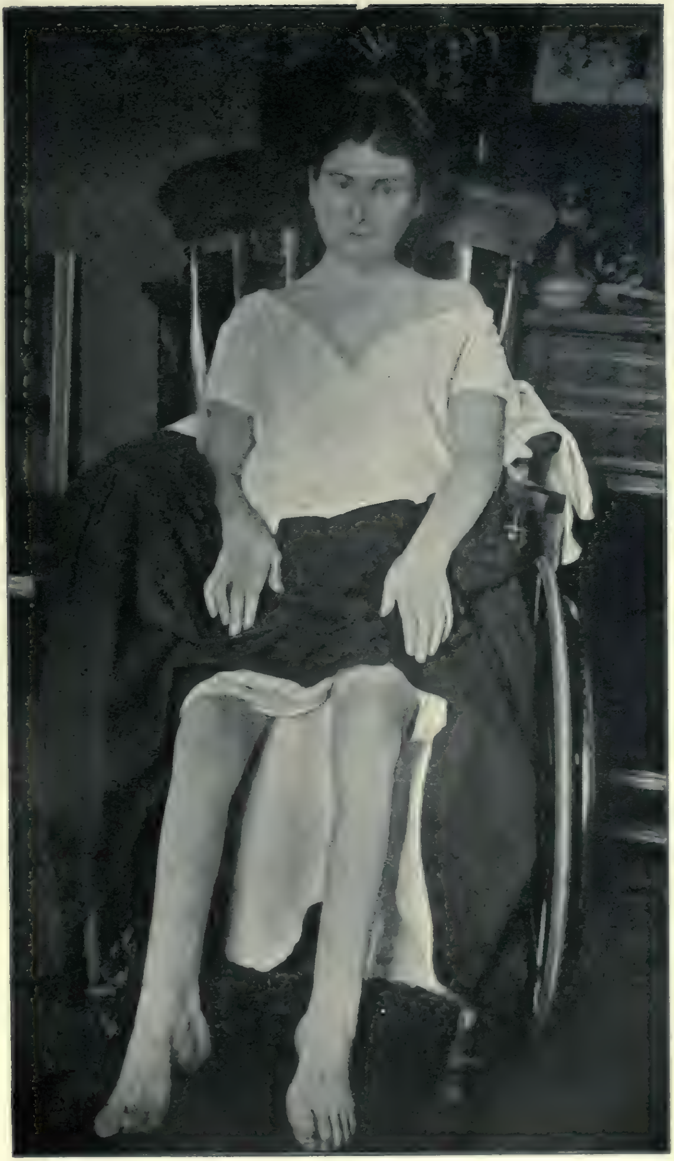

FIG. 4.

Case III.—To show *main en griffe* and deformity of lower limbs.

into line, but not flexed. The other toes show slight over-extension at the metacarpo-phalangeal joints; at the inter-phalangeal joints there is flexion. The dorsum of the foot is prominent.

Motor Functions.—Patient shows distinct weakness in the muscles that raise the *trunk* into a sitting posture. She can raise herself, but awkwardly and slowly. When sitting, there are constant swaying movements of the head and body; there are, besides, frequent jerkings of the head, and also choreic twitchings of the face, especially in the right levator anguli oris. These are not by any means so marked as to cause any confusion with true chorea.

In the *upper limbs*, movements are free at the shoulder, elbow, and wrist joints (with hand closed). When told to extend the finger and hand there is a marked imperfection; the wrist is somewhat flexed forwards, and to the ulnar side, the fingers are over-extended at the metacarpo-phalangeal joints (especially the ring and the little fingers), and flexed at the first inter-phalangeal joints. The terminal phalanges are in line with the middle. The deformity is, in short, the *main en griffe*. The thumbs are unaffected; the fingers can be separated, though less freely than normal.

Ataxia is present in the upper limbs, but patient can button her clothes, or thread a needle, if the elbows are supported. Her movement is like her sister's (Case I.) in picking up small objects, and in carrying out any movement oscillatory jerkings are very noticeable. The ataxia is brought out by the usual tests of bringing the fore-fingers together, or touching the point of her nose, with eyes shut. Hesitation and waving movement are more marked when she is near the point aimed at. The super-added movements are less marked if the eyes are open.

Power of grasping is still fair, though awkward from the extensor wasting. At elbow and shoulder muscular power is well preserved.

Lower Limbs.—Patient can flex and extend at hips and knees, but with lessened power. With her legs extended she can raise the right foot two inches off the bed, the left twelve. Even when supported on each side she cannot walk; her feet are pushed too far forward, so that she is unable to get her balance. Her steps are very short, the right foot is crossed over the dorsum of the left, and thrown two or three inches beyond the place where it ultimately touches the floor. She puts down her feet with a slight stamp, first the toes and then the heels. There is only very slight voluntary movement at the ankle, and that is mainly extension. The toes allow of somewhat further voluntary movement. The ankle can be passively moved to a much greater extent, but dorsi-flexion of the foot is much interfered with, through contracture of the tendo-Achillis. The heel can be placed on the opposite knee, but slowly.

Sensory Functions. — Occasional headaches, no giddiness. There is some slight delay in conduction of impressions from the lower extremities, but sensibility in its different modes is essentially normal. There is a small area on the front part of the dorsum of the left foot, which shows slight dulling of sensibility to touch, not to pain.

Reflexes.—No elbow or knee jerk, abdominal reflexes well marked, plantar exaggerated, no disturbance of visceral reflexes.

Eyes.—Reaction of pupils normal; when looking to either side some nystagmus-like jerkings occur—about seven in five seconds, irregular in time. They are also seen when she looks up or down; sight is normal.

Hearing.—The hearing distance varies, but is usually about three inches on each side (*watch test*).

The Tongue, when protruded, is constantly in a state of movement—not the irregular jerks of chorea, but a fine oscillatory movement.

Speech.—Her voice is low-pitched, not harsh, monotonous. She keeps her teeth rather close in speaking. Some words are slurred—" docr" for " doctor," " I don't emember" for " remember." The speech is consequently somewhat slipshod; it is very like her brother Hugh's.

Vasomotor System.—Feet tend to be cold; psychical condition normal; muscular sense normal.

Electrical reactions. Faradic current much better borne on legs than normally; somewhat better on arms. Muscles of arms contract well under its influence, but the interossei act sluggishly.

Galvanic current.—In flexors of right forearm KCC 6, ACC 7; in extensors of right forearm KCC 6, ACC 7, KOC 8, AOC 8; in antero-lateral muscles of right leg KCC 6, ACC 10. The interossei, especially those of the third and fourth spaces, respond feebly to galvanism.

B. AN "ISOLATED" CASE OF FRIEDREICH'S ATAXIA.

Case IV.—William M., aged 13, admitted to Dundee Royal Infirmary, August 30, 1894.

Family History, &c.—Patient is third of a family of four, the

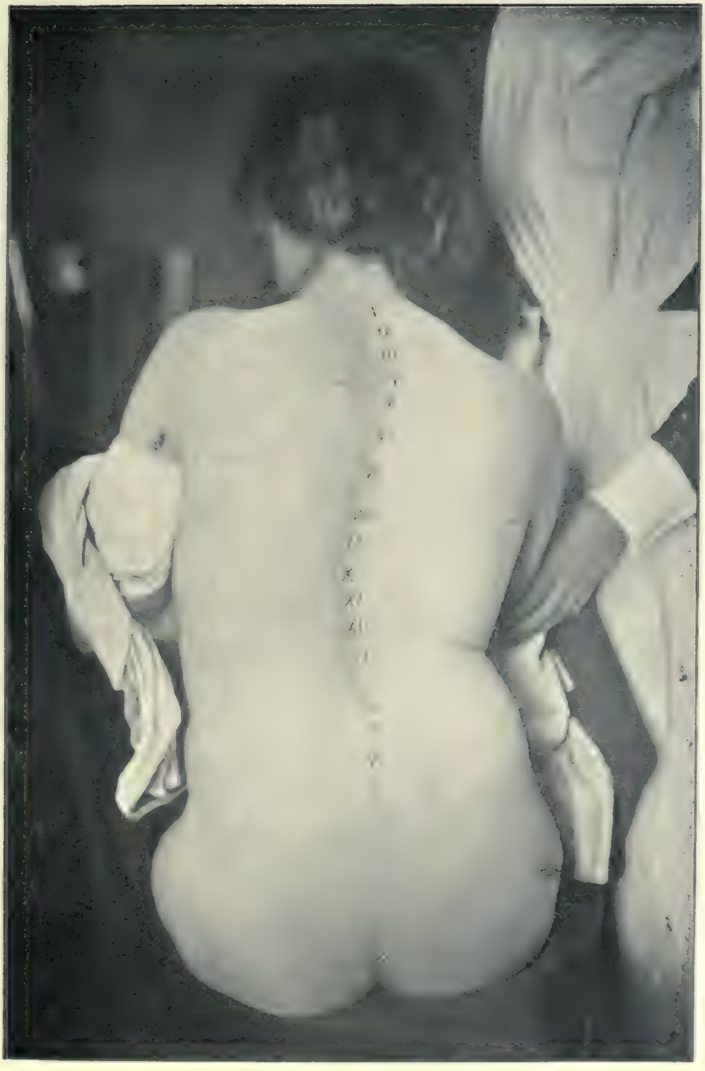

FIG. 5.

Case III.—Scoliosis. Patient requires to be supported.

youngest three by the same mother. His father died some years ago of inflammation of the lungs, after eight days' illness. His mother, sisters, younger brother, and nephew are all healthy. A slight nystagmus can be detected on extreme lateral deviation of eyeballs in his brother and younger sister. There is no history of any nervous affection in any of the other members of the family, nor is there any evidence of syphilitic or tubercular taint. His home surroundings are good; he has not been overworked at school, and has shown average intelligence.

Present Illness.—About two years ago his mother noticed that he became easily fatigued, and did not care to join in the games of his companions. His gait was a little unsteady; if he ran or walked quickly he was apt to stumble or fall. This was no worse in the dark, but if his feet were bare there was decided aggravation. There was no definite onset, no pain, shivering or feverish attack. His condition did not change much for some months, and then his upper extremities were seen to be slightly affected. He could not dress himself so quickly as usual, his hands were a little unsteady, and he was apt to spill the contents of a saucer in drinking. His handwriting was rather shaky, there was a want of freedom in using his pen or pencil and a difficulty in forming some letters. Both sides of his body seemed equally affected. The only time he complained of pain was a year ago, when his back ached at times, but this feeling passed off after the back was painted with iodine. He had no headache, or shooting pains in his limbs. During the past few months his arms and his legs have been getting thinner. No change has been noticed in his speech. There has been a steady deterioration from the beginning of the illness, the difficulty in locomotion increasing especially in trying to go downstairs.

State on Admission.—A fairly well-developed boy, with florid complexion, sad or pensive expression; brow transversely wrinkled, eyebrows often involuntarily drawn up while under observation. Weight 5 stone.

Nervous System (the only system affected).—The boy is quite intelligent, and has a keen sense of his unfortunate condition.

Motor Functions.—Patient stands with his feet about twelve inches apart, but if his heels are brought together he sways from side to side, and would fall if unsupported. Head and upper part of body bent forward. Closure of eyes increases the unsteadiness; when sitting in bed he has difficulty in balancing himself. Various muscles are seen to be constantly working as he tries to steady himself when sitting or standing. When laid on his back he can raise himself up fairly easily by means of the trunk muscles alone. When standing he can stoop forwards, and pick any article off the floor.

Gait.—He walks with head bent forwards, and eyes fixed on the ground, his legs widely straddling. He brings his feet down heavily with a slap, first the toes, then the heels, reeling from side to side as he goes, and swaying his arms irregularly. His eyes are a great help to him in walking. He walks backwards with great difficulty; it is impossible to get him to keep a straight line. His gait has something of a festinant character; his steps are irregular in length, force, and direction.

Co-ordination, as tested by making him bring the tips of his fingers together, or touch other parts of his body, or place the heel of one foot on the opposite knee, is fairly good.

Writing is shaky; he presses his hands heavily on the paper. Nutrition of muscles, good; no hypertrophy or atrophy; no true fibrillar contractions, though muscular twitchings are noticed here and there even when he is lying at rest. No deformities about feet, but slight lateral curvature of spine, dorsal convexity and lumbar concavity to left.

Speech is low-toned and somewhat slow, not jerky nor scanning nor monotonous. The muscles of his lips seem slightly weak in their action, but there is no paralysis of the facial muscles.

There is distinct nystagmus (better marked on some days than others), brought out when the eyes are fixed on an object away from central line of vision.

Reflexes.—Patellar reflexes gone; no ankle clonus; plantar reflexes present on both sides, rather better marked on left; epigastric and cremasteric reflexes present; visceral reflexes normal.

Sensory Functions.—Has no abnormal sensations such as cold, heat, numbness or prickling. Sensibility to touch seems a little dull in lower limbs, the light touch of the head of a pin being frequently unfelt. Sensibility to pain acute, can readily localise a pin prick, can easily distinguish heat and cold.

Sight good; pupils equal, regular, reacting freely to light, but somewhat slightly to accommodation.

December 13, 1894.—Tongue thick and flabby, constant tremors in it, especially on protrusion. Frequent involuntary jerkings occur in head and upper limbs. Complains of feeling weaker, and often lies in bed for a day. Has occasionally been troubled with cramp in muscles of calf. His walking and general power of balancing are much worse, and he often falls. In trying to touch an object he is more uncertain in his aim, from the irregular jerkings in his arms during voluntary movement. There is a corresponding difficulty in carrying out the heel-on-knee manœuvre.

Fundi of eyes show nothing abnormal. (Dr. Macgillivray.)

February 19, 1897.—Since last note patient has had fair general health. He has grown much taller, but there is no improvement as regards any of the symptoms described above. His speech is rather worse, the lips still seem somewhat at fault. The voice is more monotonous, and is low in pitch, distinct enough when speaking to me, but his mother says that when he is reading aloud to her, as he is fond of doing, she often has difficulty in understanding him. There is a thickness, a want of crispness about his utterance.

The ataxia of the hands is still as marked, if not more so. He can put in buttons, and write his name—always with the same awkward procedure. The superadded movements in using his hands are much as before.

He can walk, but only with strong support. His soles feel the ground quite well: the feet are raised higher than normal and brought down with a stamp, paretic not spastic. Romberg's sign is present.

When in bed he lies on his back; he has often a painful cramp, which causes the left leg to draw up of itself. The pain is immediately above and below the knee. The limb has first to be rubbed over the knee and then pushed down. If it were simply pulled down, the pain would be severe.

In the morning he has some pain in the lumbar region, when bending to get on his clothes with his mother's help. This is probably due to some stiffness, developed through his position in sleep. His back is very weak, and shows still a slight scoliosis; this is not of a permanent character.

His favourite position is sitting on the floor, with his back against the wall.

A recent symptom is a difficulty in commencing the act of micturition; there is no dribbling afterwards.

A brief *résumé* of these cases will be useful, and will show the main diagnostic points in Friedreich's ataxia.*

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* (I take this opportunity of acknowledging the valuable aid given me in taking Cases I. and IV., by Dr. C. Kerr, then house physician in Dundee Infirmary, and by Dr. A. Croll, resident physician in the Parochial Hospital, in recording Case III.

REMARKS ON SOME OF THE IMPORTANT SYMPTOMS.

Disturbances of the motor system are by far the most important in the symptomatology of Friedreich's ataxia. So much is implied in the name, and Friedreich himself pointed out that the ataxia appears in two forms—ataxia or voluntary movement, and static ataxia. Choreiform jerks, nystagmus, tremors of tongue, and possibly disturbances of speech, are further evidence of wide-reaching motor invasion, while in the later stages of the disease certain muscles are apt to become paretic or paralysed.

Ataxia of voluntary movement is well shown in the gait. It has been often described, and has a character which marks it off from that of tabes dorsalis. In two of my cases (Cases I. and IV.) alone could it be examined. The patient in walking keeps his legs apart, and has difficulty in keeping to a line. He reels a little, sometimes a good deal, like a drunken person. The feet are brought down with a slight slap, first the toes and then the heels. The steps are short and vary in their length. The arms sway to some extent to aid in balancing, and for the same reason the eyes are fixed on the ground, a little in front; the head is bent forwards. In tabes the movements of the limbs are more disorderly, there is more excess of range, more exaggeration; the foot is raised higher, and brought down with more noise. The patient can keep to a line, if not asked to put one foot directly in front of the other. The character of the gait in each case varies, however, according to the stage of the disease, and, in tabes, according to the muscles worst affected. The point about the gait of Friedreich's ataxics which is most important from a diagnostic point of view, is its cerebellar character. This occurs also in Marie's disease, in multiple sclerosis ("rarely," Leyden), and of course in cerebellar disease.

But, added to the cerebellar or tabeto-cerebellar character, are various movements of the limbs and trunk best studied when the patient stands. These movements come under Friedreich's designation of *static ataxia*.* They are perhaps

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* In one of Senator's cases (34) examined when the disease had made only very slight progress, the locomotor ataxia was very slightly marked, while the static ataxia was pronounced.

CASE I.	CASE II.	CASE III.	CASE IV.
(1) Family character.	(1) Family character.	(1) Family character.	(1) No family character.
(2) Onset soon after a fever, possibly typhoid, when 7 years old.	(2) Severe cerebral affection in infancy. Onset of present illness at 12.	(2) Acute illness (? meningitis) at 15, immediately followed by symptoms.	(2) Onset, insidious at 11.
(3) First symptom, difficulty in walking.	(3) First symptom, unsteadiness in walking.	(3) First symptom doubtful.	(3) First symptom, affection of gait.
(4) Very slow advance of ataxia; cerebellar character, slightly marked; worse in the dark (Romberg's symptom).	(4) Advance of ataxia, with remission, between 17 and 19; again rapid advance, with paralysis of legs at 20.	(4) Ataxia of gait, gradually increasing. Paraplegia nearly complete at 26.	(4) Ataxia of legs (tabeto-cerebellar), going on to complete inability to walk alone. Static ataxia. Romberg's symptom.
(5) Slight change in speech, beginning somewhat early.	(5) Marked articulatory defect.	(5) Articulatory defect.	(5) Speech only slightly affected and that recently.
(6) Slowly increasing ataxia of upper extremities.	(6) Ataxia of hands at 13.	(6) Ataxia of hands since the age of 15.	(6) Ataxia of hands and fingers, noticed after some months' affection of legs.
(7) Swaying of body; choreiform jerks.	(7) Choreiform jerks and swaying.	(7) Choreiform jerks and swaying.	(7) Constant swaying of body. Choreiform jerks.
(8) Nystagmus.	(8) Nystagmus.	(8) Nystagmus.	(8) Nystagmus.
(9) Absence of knee-jerks.	(9) Absence of knee-jerks.	(9) Absence of knee-jerks.	(9) Absence of knee-jerks.
(10) Slight weakness of interossei in hands, slight scoliosis. No talipes, or hyperextension of great toe, but unusually high instep.	(10) *Main en griffe* on extension of fingers. Kypho-scoliosis, very marked. Talipes equino-varus.	(10) *Main en griffe*. Kypho-scoliosis. Talipes equino-varus.	(10) No deformity of hands or feet. No permanent scoliosis.
(11) No marked paresis unless in hands.	(11) Great loss of power in arms, trunk, and especially in legs.	(11) Paretic condition in back and legs.	(11) Weakness of muscles, especially in back and legs; no atrophies.
(12) Intelligence and sensory functions (general and special) normal. Perhaps some impairment of muscular sense.	(12) Intelligence normal. General and special sensory functions normal. Muscular sense affected.	(12) Intelligence normal. Sensory functions normal. Muscular sense normal.	(12) Intelligence normal. Sensory functions normal.
(13) Visceral functions normal. Pupil reflexes normal.	(13) Delay in commencing act of micturition since age of 20; visceral reflexes otherwise normal. Pupil reflexes normal.	(13) Visceral functions and menstruation normal. Pupil reflexes unaffected.	(13) Visceral reflexes normal, till quite recently, when a difficulty in beginning the act of micturition came on (as in case II.). Pupil reflexes unaffected.

most strikingly shown when one looks through the camera at the patient. The inverted image is seen to be in a constant state of unrest, from swaying movements which are in inverse proportion to the amount of support afforded to the body. When patient lies in bed they are absent; when sitting with the back well supported by pillows, the head and neck move; if sitting unsupported, the movements are more marked, as the trunk is now swaying; and when standing unsupported, they may be seen at their maximum. A rough examination, then, shows the patient to be standing with legs apart, head and upper part of body bent forwards, tendons about ankle and on dorsum of feet in a state of perpetual motion, toes often with an athetoid movement, grasping the floor.

A more careful examination may show the various muscles of thighs, hips, and back in a state of irregular contraction, in the endeavour to preserve equilibrium.

Ataxia of movement is shown in the lower limbs when patient is lying by such common proceedings as putting one heel on the other knee. This test is carried out by Cases I. and II. very well, much better than one would expect.

Ataxia of upper limbs is easily brought out by writing, threading a needle, putting in buttons, picking up a small object.

The super-added oscillatory movements are present in carrying out any such attempt, and the movements are clumsy and awkward. The patient when lying seems to have more control over the test movements of the feet than of the hands, possibly because those of the latter are more delicate. Senator[35] notes that in his first case, incoordination is less marked in the lower extremities when lying or walking, than in the upper.

Case I. illustrates the bad effect of rest, for she is always worse as regards the ataxia in the morning, quite apart from the degree of light.

Tresidder[38] notes the same to a much greater degree in his Case I. He says "When 23 years of age, the unsteadiness in walking was so pronounced that a London physician was consulted, who thought that the lateral curvature of the spine was producing paralysis, and the patient was ordered to lie down continuously. She did so for thirteen months. After this she began to walk again, and was at first very weak, but in the course of some months regained her strength until she could walk nearly as well as before the thirteen months rest."

Besold's second case,[54] a hawker, believed that his long walks (20-25 kilometers) were of great service to him.

These facts are of interest and importance, in view of the excellent results in the mechanical and gymnastic treatment of tabes attained by Leyden, Frenkel and others.

Organic changes are not affected, but the function of the muscles is improved by this method of "compensation-therapeutics." (Leyden and Goldscheider, Erkr. d. Rückenm, page 589.)

The test of making the patient close the eyes when standing or walking (Romberg's sign), while of very great value in locomotor ataxia, is not so useful in Friedreich's disease. Increase of unsteadiness through this is often absent, as in three out of four of Friedreich's own cases, in Rosenbaum's[83] elder case, in Bramwell's[12] first case. In my own cases that can walk (I., IV.) there is no doubt about the existence of this sign. In Senator's first case it was better marked on the second examination than on the first, a year and a half intervening between the two.

Where the patient can no longer walk, as in Case III., the same test can be applied to the upper extremities; here there is increase in the superadded movements when the patient is using her arms, with eyes shut.

The so-called *nystagmus* of Friedreich's disease seems best classed with static ataxia. It is not a true rhythmical nystagmus, in the sense of the ophthalmologist, though this occurs in rare cases (noted in three of those analysed by Griffith). The symptom must be carefully looked for, and brought out by directing the patient's gaze to one or other side. When the muscles of the eye-balls have their tension thus increased, distinct jerkings are seen—slow they may be and irregular, as in Case III., when the rate was seven in five seconds. Sometimes the movements can only be elicited by turning the patients three or four times round, as in Geigel's[28] two cases (brothers, ages 20 and 18). On turning the patients to the right the eyeballs both turned to the right, and the short jerking movements of nystagmus to the left followed, lasting a quarter of a minute.

In two of Mendel's[10] cases, the movements thus brought out were very marked, and lasted some minutes.

They are found in most cases of Friedreich's disease—(" not in all " Gowers), but they are likewise often found in persons who have weak health, and even in the perfectly healthy.

This nystagmus I found in two quite healthy members in each of the families to which my cases belong, but no pathognomonic stress is to be attached to this.

On Schultze's instigation, Ausschlag[53] examined 200 unselected cases, and of these only 25 showed no tremor, though frequently there was only a single movement too far. " Only a strikingly high degree and special frequency of movements can be regarded as pathological."

Even modified nystagmus (" nystagmoid jerkings) is very uncommon in locomotor ataxia, so this sign may in certain cases be a help in diagnosis.

The involuntary movements, like an irregular tremor shown in the protruded tongue, may, without doubt, be referred to the same category as those we have been considering, and some would similarly treat the speech disturbances.

Besides the swaying movements of static ataxia, there are frequent *involuntary jerkings, choreiform in character*, but not so constant or violent as in chorea.

Thus in Case I., occasional twitchings are seen in the face, especially drawing up the right angle of the mouth. Very much the same facial movement is seen in her sister.

In Case IV., the brow is often raised involuntarily. The transverse wrinkles so marked in his brow may be associated with the bent position of his head, and may have been produced by his raising his eyebrows along with his upper eyelids. A waggling or wobbling of the head is constantly to be seen in the two worst cases, and to a slighter degree in the others. Even when lying at rest occasional muscular twitchings, not fibrillar, are seen in Case IV. He is subject to a cramp in the left leg at night.

The other three cases have all been subject to jerkings in the legs at night. In the case of the eldest (Case II)., they seem to be more of the nature of cramps now, and another member of the family has often to get up to pull his legs straight.

The choreiform jerkings are an almost invariable part of the clinical picture of Friedreich's ataxia, and are of great diagnostic importance. They have sometimes led to a false diagnosis of chorea, as will be noted later on (*v.* Diagnosis).

In one case (Zabludowski's[84]) are noted "choreiform movements, especially when going to sleep," and also at that time, twitching in face and ears, and flexion of thighs on abdomen.

Painful cramp like contractions when in bed have been noted by others (Bramwell,[13] *Atlas*, vol. i., p. 46; Besold [54] Case II.). Nocturnal restlessness is noted in Bramwell's [12] Case I. Some of these exaggerated movements at night are probably analogous to the jerks which most people experience on dropping off to sleep.

Athetoid movements occur, too, sometimes in a quite minor form, as in one of Griffith's cases where was no tremor but a slow ataxic movement, resembling athetosis, which appeared when the hands were lying passively in the lap.

Athetoid movements of the toes in walking are occasionally present (Ladame's case,[1] p. 477). In both of Wallace Anderson's [36] cases, athetosis was distinctly marked in fingers and toes. These cases were peculiar in other ways; the eldest was twelve years old, but looked six or seven, and his mental condition corresponded to this age. The knee-jerks were present, though faint in both, and so was rigidity of legs with spastic gait. The onset in both was after measles two years previously.

The most remarkable movements of this class were observed in three cases reported from France. The first

(Chauffard[33]) was a boy of eight; symptoms since age of three; no heredity, sisters unaffected. No nystagmus, gait neither cerebellar nor tabetic. One leg made to pass before the other with a jerky and exaggerated convergent movement. Among usual symptoms were the progress, absence of jerks, absence of ocular symptoms, comparative preservation of muscular force, slight equino-varus. Further there was marked lordosis (dorso-lumbar), with slight cervico-dorsal kypho-scoliosis. No choreiform instability in repose, but sudden involuntary flexions of lower extremities at hips, legs remaining extended. Intelligence good, no grimaces. "Hovering" inco-ordination in picking things up. Arms took up athetoid position in walking with support. Forearms in position of pronation, hands flexed, their backs being directed down, fingers extended or flexed in the palm. The movements were not the usual slow convulsive movements of double athetosis. They only appeared when other voluntary movements were made—a muscular synergy, an athetoid synkinesia.

Chauffard easily separates this case not only from athetosis, but from tabes, insular sclerosis, cerebral diplegias, and chorea. But though there are several points of contact with Friedreich's ataxia, it is difficult to get over the uncommon features. The other two cases, reported by Londe and Lagrange,[72] are more like the ordinary type: Two sisters, aged 15 and 16, both took ill at six or seven. At first they were regarded as chorea. Absence of jerks, hollow-foot, marked inco-ordination, no nystagmus, internal strabismus in both; gait staggering; involuntary facial movement; head continually moving in various directions; choreiform jerks in head and limbs; *danse des tendons* in standing; slight scoliosis, no sensory or sphincter troubles, no affection of pupils or fundi; mental dulness. Athetoid attitude exactly as in Chauffard's case.

It is curious that in no one of these three cases was nystagmus present; in the latter two the internal strabismus, the mental dulness, and the athetoid attitude confer a special family mark.

Deformities of extremities and spine.—Certain deformities affecting the feet and spine have been always regarded as characteristic of Friedreich's ataxia. To these must be added the affection of the hand shown in my first three cases. None of these conditions is essential; the talipes (equinus or equino-varus) was noted by Griffith in only 27 cases out of 143. In a number of instances besides these there was dorsal flexion of all or some of the toes. The latter condition in itself might, as Besold suggests, be attributable to "modernes Schuhwerk," unless uncommonly well-marked. In Rütimeyer's families, Blattner and Kern, early prominence of the extensor propr. hallucis was regarded as a sign of very bad augury—it was one of the first symptoms (Ladame). Baskett's case,[76] a girl of 13½, was treated at a hospital for double talipes varus at the age of 8½. Her symptoms began when she was six, but usually deformity of the foot is of late occurrence. It is well-marked in Cases II. and III., but not in the other two. In Case I., however, there are indications in the high instep and occasional dorsi-flexion of the great toe that the shape of her foot will become like her brother's and sister's. The general appearance in a marked case corresponds closely to *pes cavus*, "hollow claw foot." The claw position may or may not be assumed by the terminal phalanx of the great toe. In Case II. (photo) the right great toe is over-extended as regards both phalanges. This is so, too, in Case III. In Plate X. in *Med. Annual* for 1895, representing a case under the care of Mackay, of Devizes, the great toes, like the others, have the second phalanges flexed, and this seems to have been the way in Ladame's patient.

Dreschfeld[50] describes a case which showed no talipes, but hyper-extension of the great toes "resembling that seen in the early stages of peripheral neuritis."

Deformities of the hands have been rarely observed. Bramwell[23] gives a family of three showing *main en griffe*. Griffith (*Amer. Journ. of Med. Sciences*, 1888, p. 377) describes one of his own cases as having "grasps strong but claw-like," another as having claw-like hands; but he may be simply referring to the mode of prehension so common in Friedreich's ataxia. In two of Rook's[7] cases, the hand was claw-like and one had wrist-drop. One of Taylor's[55] cases had a little difficulty in extending the fingers.

In Cases I., II., and III., the attempt to extend fingers in line with the forearm at once brings out a more or less pronounced *main en griffe* (*see* fig. 3). Let me recapitulate the description of the condition in this case. When the hand is lying passively, nothing wrong is noticed. When he is told to straighten his fingers, flexion is produced at the wrist, with some bending towards ulnar side. The near phalanges are over-extended in the four inner digits, more so in the fifth than the fourth, in the fourth than the third, and so on. The middle phalanges are flexed, and the terminals are nearly in line with these. In regard to these joints, too, the deformity is more marked in the inner than in the outer digits. The fingers are separated a little from one another. The thumb is simply bent back. The more strongly patient tries to straighten the fingers, the greater does the alteration in form become. The extensor com. dig. tendons can be seen standing out on the back of the hand, and can be traced up the lower third of the forearm. The palmaris longus tendon, too, stands out prominently. The fingers can be adducted and abducted, but to a much less degree than normal.

What is the mechanism of this condition? It has nothing to do with the proper flexors and extensors of the wrist, for when the hand is closed, movement may occur at the wrist in the normal four directions—flexion, extension, ab- and adduction. It is not a paresis of the extensor com. digitorum; its tendons are inserted mainly into the near ends of the near phalanges, and Duchenne hence says they might be called *extensors of the near phalanges*. In the above case, so far from there being any sign of their failure to act, the chief trouble would appear to be their over-action (over-extension of proximal phalanges).

The cause is, I think, a paresis of the interossei—not paralysis—though the condition seems to come nearer this the older the patient becomes (compare condition in the three first cases). The interossei flex the first phalanges, and extend the second and third; they also abduct and adduct the fingers. When they are weakened, and when the common extensor is thrown into action, the fingers assume the position above described. To keep the first phalanx in line with the metacarpals, antagonistic action of its extensor and flexor is needed. "When the interossei fail, the unopposed extensor causes an exaggerated extension of the near phalanges, and the extension of the middle and far phalanges does not only not occur, but they are, on the contrary, flexed in direct proportion to the degree of extension of the near phalanges." (Duchenne, *Syd. Soc. Trans.*, p. 265.)

The flexion at the wrist may be thus explained: when the hand is extended, not only do the proper muscles of the wrist act together, but also the long extensors and flexors of the fingers. The combined effect of their synergic action is to place the metacarp in line with the forearm. If the interossei are weakened, and the near phalanges are overextended, the balance is upset, the common extensor acts at a disadvantage as regards its extending action at the wrist, and there is the appearance of a slight drop-wrist. This can be imitated to some degree in a healthy limb.

The greater amount of distortion in the ring and ulnar fingers corresponds to the condition in injury of the ulnar nerve, and is accounted for in this case by the escape of the first two lumbricals, which are supplied by a branch of the median. That the whole terminal part of the ulnar is not affected is proved by the intact sensibility, among other points.

The simpler case here considered leads to the consideration of the "Friedreich foot," about which a good deal has been written. I believe that it is analogous to the condition just described, and that its formation can be referred to an affection of the interossei of the foot (including the short flexor and adductor of the great toe). There may also be a real weakening of the antero-lateral muscles of the leg; there is certainly some retraction of the calf muscles, and tightening of the tendon-Achillis.

Paretic condition of various muscles besides the interossei is present in advanced cases. This is shown in the helplessness of Cases II. and III., especially as regards the whole of the lower extremities. The muscles that act on the spine may also be involved. In early cases, decided loss of power is usually absent.

Curvature of the Spine is very common in Friedreich's ataxia. Soca, quoted by Hallion,[24] says it occurs in half the cases. The photos of Cases II. and III. show the usual marked conditions. In the latter, the main convex curve is to the left, and is greatest about the ninth dorsal vertebra; in the former it is to the other side; it is both longer and more marked, and it is worst at the sixth or seventh dorsal vertebra. Perhaps the convexity to the *left* in the young woman is associated causally with the greater weakness in her right arm from her acute illness. In the common lateral curvature of adolescents, the main dorsal convexity is usually an exaggeration of the natural curve to the right, as in the brother (Case II.). In both there is kyphosis in the cervico-dorsal region, in the man this being so marked that the vertebral spines are nearly horizontal when he is sitting up. Observe how close the numbers on the spines appear to be, the effect of photographic distortion (fig. 2).

Hallion[24] has made a careful study of the spinal curvatures in nervous diseases, especially syringo-myelia, infantile paralysis, Friedreich's disease and locomotor ataxia. In all, the gray substance of the cord is affected in a different degree: in Friedreich's disease a pathological change is found in Clarke's columns, where some localise vaso-motor centres. Have we here, too, he asks, nervous lesions capable of causing an alteration of the bones, and specially of the vertebræ? He quotes Soca, to the effect that muscular weakness alone is sufficient to account for the condition. The want of tone may involve ligaments and bones too, as in the common scoliosis, but of this there is no proof.

We do not require to postulate any marked paresis of the spinal muscles; adolescents, who are subjects of the ordinary lateral curvature, are fairly muscular. A want of balance about the groups of muscles is more likely to start the scoliosis.

REMARKS ON THE CONDITION OF THE SENSORY SYSTEM.

The *sensibility of the skin* is frequently quite normal as regards pain, touch and temperature, but on the other hand it is not uncommon to find a slight dulling. In Cases I. and IV. the condition is practically normal; in Case III. there is some delay in recognising touch in the lower extremities, but localisation is correct. At one time there was absence of sensibility to touch on the dorsum of the left foot and the middle toes.

Case II. shows slight diminution of acuteness in recognising touch in the legs, but pain is felt normally; in the upper extremities sensibility to touch is normal. The curious thing about his condition is, that at one time (1884 and earlier) there was decided anæsthesia to touch and pain below the level of the iliac crest, this being proved by the examination in the Infirmary (1884), and also by the fact that about the age of twenty (1883) he burnt his foot by standing on a hot coal without feeling any pain.

It is interesting to compare this analgesia with the condition in the three cases recorded by Klippel and Durante, quoted in Londe's Thesis.[61] These cases are amongst the most important selected by Marie, from which to build up his type *hérédo-ataxie cérébelleuse*, but they are distinguished from the rest of this group, as well as from ordinary Friedreich's ataxics, by the disturbances of sensibility.

In Louis H., in 1892, tactile sensibility was abolished in legs and feet, much diminished in forearms and hands, and diminished in face; no plantar reflex; sensibility to heat recognised everywhere; retraction of field of vision. In 1894, Londe found analgesia in hands and legs; sensibility to touch present; sensibility to heat and cold weakened in the analgesic parts, retraction of field of vision hardly perceptible.

In François, sensibility to touch and cold, abolished below the knee in 1892, two years later no affections of sensibility.

In their sister, complete anæsthesia to all forms of sensibility on the internal aspect of the leg, in 1888; whereas a few years later, sensibility in her too, was normal.

Londe gives various reasons for his opinion that these sensory disturbances were due to hysteria. Certainly one is justified in regarding the condition as functional, and it has to be remembered, that many of the symptoms in nervous diseases, which have a well-marked organic foundation, are in all probability purely functional.

One of Stintzing's cases (*Münch. Med. Woch.*, No. 21, 1887) had complete anæsthesia in the lower extremities, and Senator's [35] second case felt on the feet needle pricks and differences of temperature, only slightly or not at all.

Déjerine [4] describes a brother and sister who were affected with marked sensory troubles of the extremities, diminishing towards the trunk, and also corresponding muscular atrophies; these he regards as true cases of Friedreich's ataxia, with very marked exaggeration of certain symptoms, sometimes met with in this disease, though only to a very slight degree. He refers them to lesions of the peripheral nerves and sensory roots.

It is interesting to note that Risien Russell, by his recent experiments (*Trans. Phil. Soc.*), showed that anæsthesia and analgesia occur in the partially paralysed lower extremities, after removal of the cerebellum.

Subjective sensations are also occasionally found. In Case II. these used to be, formication in the soles of the feet, and tingling sensations in the legs. Case I. had for a short time pains in the back of the thighs.

Lightning pains occurred in one of Déjerine's cases, very much the same as in locomotor ataxia. Bramwell [13] reports a family group of three cases, all of whom suffered from lightning pains at one time or another, one from gastric crises and all from exaggerated sensibility. As he notes, one of Charcot's showed lightning pains.

Other cases have involuntary spasms, associated with itching or other sensations (Tresidder's).

MENTAL CONDITION.

A normal mental condition may still be looked upon as characteristic. Tresidder's [38] three cases showed remarkably high intelligence, one of them having taken an honours degree at Cambridge. Ladame's[1] case had a very vigorous memory and intellect. Hector Mackenzie's[48] was bright and intelligent for his station in life. My own cases are not affected mentally in the least degree. The *expression* of face is often unintelligent (as in Case IV.), and likely to mislead the observer. Cases that have normal mental power may be easily excited to uncontrollable laughter (Brock,[41] Ladame, Bramwell).

Mental defect is noted in some cases. In one case, epileptic attacks and dementia (Hoffmann, of Heidelberg[71]); in another imbecility (Power, quoted by Gowers, *St. Bart.'s Hosp. Reports*, 1882, p. 305); in two sisters, marked mental defect (Londe and Lagrange[72]); in one of Taylor's[55] increased mental irritability, with pronounced feebleness of intellect; in the other, not related to the first, epileptic attacks; in Nolan's[63] three cases, congenital idiocy; in Vinaj's[69] mental degeneracy.

In some of these, however, the diagnosis is more than doubtful. In itself it would not be surprising to find mental defect added to the usual symptoms; it would represent a further stage of the neuropathic tendency.

An American professor (Halbert,[68] Hahnemann Hospital, Chicago) gives a report of two cases of inherited syphilis with spastic-paralytic symptoms, as cases of Friedreich's ataxia, and in regard to the elder—eleven years of age—makes the quaint observation that "his mental condition is not greatly impaired, except that it is infantile from the lack of development!"

More will be found on this subject of mental condition under the heading of diagnosis (page 105).

REFLEXES.

The condition of the *superficial reflexes* in Friedreich's ataxia may be regarded as normal.

In Case II. plantar reflexes are well marked on the right, slightly on the left. The cremasteric and abdominal reflexes are well marked. In Case III. all are well marked, and the plantar exaggerated. In Cases I. and IV. the superficial reflexes are normal.

In Berdez's [79] case, superficial reflexes were very lively. In three of Besold's cases [54] they were normal, in the fourth, abdominal reflexes were absent.

Variations in degree of cutaneous reflexes are of course of constant occurrence in healthy persons, and the point under discussion is chiefly of importance as helping to distinguish Friedreich's ataxia from tabes. In the latter affection the plantar reflexes, especially, are often absent, this being due to the loss of cutaneous sensibility.

Much more important is the *condition of the deep reflexes*, of which the knee-jerk alone may be considered here, as it is easily tested.

In the four cases I have described the knee-jerks are completely absent, and this is the case in the great majority of examples of Friedreich's ataxia.

Crozer Griffith (*loc. cit.*), in his analysis of a hundred and forty-three cases, describes the abolition of the knee reflexes as very early and very constant. In ninety cases only was their condition mentioned, and curiously enough in as large a proportion as twenty-two of these they were present, in six being actually exaggerated. It is true that Griffith's statistics are compiled from an unsifted mass of cases, but one can certainly not go as far as Ladame, who holds that the abolition of patellar reflexes is an absolute rule in this disease, and that one would do right to mistrust the diagnosis of Friedreich's ataxia whenever the knee reflexes are not lost, although all the other symptoms would seem to confirm it (BRAIN, vol. xiii., 1890, p. 492).

When one comes across a typical family of Friedreich's ataxics, such as that described by Tresidder,[38] and finds the knee-jerks completely absent in one and present in the others, one is forced to the conclusion that this test is not infallible. Moreover, it seems probable that the disappearance of the knee-jerks is, sometimes at least, not one of the earliest symptoms.

In Senator's [34] [35] first case, these were found to be simply weakened one year after the onset of the illness. After the lapse of another period of eighteen months the right was quite gone, the left could be obtained only by

Jendrassik's method. A sister of this patient, who had been ill for twenty years, had no patellar reflexes.

Brock's[41] second case, a man aged 27, had knee-jerks, though they were absent in his ataxic brother and nephew.

Erb[9] and Mendel[10] both showed cases in 1890 with tendon reflexes, in Erb's exaggerated. In one of Mendel's the jerks were present in May, but absent in the following November.

Londe[9] would class Erb's cases with Marie's hereditary cerebellar ataxia simply on account of the exaggeration of the reflexes, though vision was normal and talipes present.

In the cases I have quoted, as in two of Bramwell's,[22] the presence of the jerks may not be sufficient to blur the picture of Friedreich's ataxia, but it is a different matter when one finds that, along with retention of knee-jerks, there are other important deviations from the type. Bouchaud,[60] for instance, records a case which differs from Friedreich's ataxia not only in retention of the jerks, but in the impaired mental condition, hallucinations, affection of vision, nature of gait, and a too-pronounced nystagmus. I might refer further to the cases reported by Wallace Anderson,[36] Vinaj,[89] one of Nolan's,[63] T. S. Wilson's[39] first and fourth, as examples that are open to the above criticism.

However, the pathological anatomy of the disease varies, as we shall see later on, to such a degree that it is easy enough to suppose the knee reflexes might be free from interference in an occasional case. Such a case might be comparatively free from sclerosis in the lumbar portion of the cord.

Of the *visceral disturbances*, that noted in Cases II. and IV., viz., slow inception of the act of micturition, is not so very infrequent (thirteen times in Griffith's analysis).

ORDER IN WHICH THE SYMPTOMS APPEAR.

This is not easy to fix, but usually the ataxia of the legs shows itself first. This seems to have been the way in all my cases; in Case I. the speech was very soon involved, but so slightly that it would have remained unobserved, but for the brother's thorough knowledge of the symptoms that might be expected.

In James's,[40] and one of Brock's[41] cases, the spinal curvature seems to have been the first symptom noticed. Both cases were lads of 18. One of Besold's[54] showed the ataxia first in the arms. In one of Mendel's,[10] the ataxia of the arms was noticed in the thirteenth year (writing becoming worse and more illegible), while it was two years later before the gait was observed to be changed. Griffith, in his analysis, states that the lower extremities were first attacked in 114 out of 143 cases, the arms in 10, and arms and speech simultaneously with the legs in 8.

MODE OF ADVANCE OF SYMPTOMS.

Some writers speak of the steady progress of the symptoms of the disease, and contrast this with the remissions of insular sclerosis. The mode of advance of the disease varies, however, to a great extent in different cases, and one may often distinguish remissions, or at least periods in which the symptoms are quite stationary.

Case II. showed slowly advancing symptoms till he was seventeen ; then for two years there seems to have been a remission. At nineteen there was rapid deterioration ; scoliosis and paresis, with unfitness for work, developed. Then the disease passed into its present chronic and almost unchanging state.

In Case III. the periods are less distinct ; for years there has been hardly any alteration, but at about the same age as in her brother's case the extreme difficulty in walking developed rather rapidly.

The other cases (I. and IV.), show a steady advance, much more rapid in the latter than in the former. The age of nineteen or twenty will probably be critical in the history of Case I.

PERIOD OF ONSET.

The date of onset of Friedreich's ataxia is by no means easy to state. Friedreich himself, judging from his own cases, considered that it was to be regarded as a disease of puberty. Most cases undoubtedly begin before this, the seventh and eighth years being most common (Gowers[88]), and it is rarely that one commences after sixteen. The onset is so gradual, unless precipitated by an acute illness, that it may be almost impossible to definitely mark it. One of the latest cases seems to be Auscher's,—the woman being twenty-five when symptoms first appeared, but as we shall see, the diagnosis of this case must be accepted with reserve.

Londe (*Thèse*, p. 154) refers to two cases lectured on by Charcot, in 1893—brothers, in whom the onset was at twenty-five and twenty-one respectively.

In my cases, the fourth took ill about the age of eleven, but the beginning was insidious.

Case I. was seven when she had the illness, which seems to have been very soon followed by slight leg symptoms.

Case II. had a prolonged illness, possibly hydrocephalus, between his third and sixth year ; there was, however, a long interval of good health before the ataxia became noticeable at the age of twelve.

Case III., like her sister, took ill immediately after a severe illness, though in this patient it seems to have affected the brain. She was then fifteen years of age.

The general statement holds good that it is a disease of the developing period.

DIAGNOSIS.

The diagnosis of Friedreich's ataxia seems to become more involved as time goes on, and cases accumulate. In typical family cases, such as the three first above recorded, there can be little difficulty if one is at all cognisant of the group of symptoms that are required to define the affection. But the symptoms are by no means few, and not infrequently some are wanting. Hence, on the one hand there are close relationships with some other nervous disorders, and on the other more or less atypical cases occur, as to whose exact position there is disagreement amongst the highest authorities. And even in regard to some cases where an autopsy has been made, and where very decided lesions have been found, there are differences of opinion; the clinical symptoms may put a case into one category, and the pathological anatomy into another.

Senator[35] considers that Friedreich's disease is a clinical conception, which would exist without any anatomical substratum, just as the notion of diabetes mellitus is independent of anatomical ideas. The pathological anatomy of some forms of diffuse myelitis ("combined sclerosis") is very like that of some cases of Friedreich's disease, but the clinical appearances are so distinct that no one could confound them.

The following case, one would almost think, had been diagnosed as Friedreich's disease after the *post-mortem* examination. It forms the basis of an elaborate paper by Tedeschi,[85] entitled "Die Friedreich'sche Krankheit—kritische und pathologisch-anatomische Untersuchung."

The patient, an orphan boy, aged 17, was admitted into the asylum at Florence in May, 1893. Two cousins in an asylum with mania. First symptom five years before, through a fright; tremor of right hand gradually involving arm, then left arm, next the legs. The tremor was like chorea. No pain; no remarkable affection of sight or hearing; no deformity. In asylum, increase of choreic movements, head involved. Walking difficult through incoordination, like an advanced tabetic. Speech slow and distinct; irritable, quarrelsome; of limited intelligence; could hardly understand what was said. Died in February, 1894, of tuberculosis.

One would like information about the knee-jerks, nystagmus, scoliosis, ataxia of upper extremities, and more about the sight and sensory system. But even with the details given—the mental affection, the uncommon onset, the asymmetry of onset, the absence of cerebellar type in the gait—I would have no hesitation in saying one does not, from the description, get the clinical idea that is requisite for the diagnosis.

The autopsy showed, besides tuberculosis in various thoracic and abdominal organs, a degenerative process in the posterior ganglia and posterior roots; marked sclerosis of the posterior columns and Lissauer's zones (reminding one in extent and arrangement of the lesions in tabes dorsalis); marked atrophy of Clarke's columns and a slight change in the direct cerebellar tract in the cervical region. The cord was strikingly small, and there was chronic lepto-meningitis spinalis, specially posteriorly. The histological examination is most carefully described, but does not help the student of Friedreich's ataxia, if the above criticism is justified.

This, like Case IV., is a so-called "isolated" case—that is, he is the only one of the family affected with the disease. We are thus deprived of one important aid in diagnosis, namely, the "family" character.

Isolated cases are found to be not uncommon, and a survey of a large number in the more recent literature makes it clear that there are no outstanding characters which distinguish them from the true family cases occurring in collaterals. Divergences from the complete clinical type occur here and there, just as they do in the family form; and just as each family affected with Friedreich's disease shows its own special peculiarity (Marie[26]), so one must make a certain allowance for individual difference in the isolated form. This much being granted, there is still room for the criticism that in some of the published cases the narrator has shown a somewhat excessive desire to fit his irregularly shaped peg in the round hole of the "clinical picture." Take for instance the personally observed case given by Dana[17] in his article on Friedreich's disease in Keating's "Cyclopædia of Children's Diseases."

A lad, 18 years old, got an injury to the back of his head five years before. Soon he began to feel dull, heavy, and drowsy, had pains about knees and ankles, severe pains in thighs, pains in lumbar region, constant and severe cephalalgia, formication in legs, back, and shoulders. Stomach symptoms, vomiting occasionally, staggering gait, ankles unsteady. Felt as if tongue were tied back, consequently speech thick. Face became and remained flushed; much dizziness; persistent polyuria (180-190 oz. daily, s.g. 1005-1010, no sugar or albumen). Diplopia.

So much for the history.

Physical examination showed a vaso-motor paresis of face and hands; "drunken" gait; no choreic or tremulous movements, except that head oscillated to and fro when he stood; slight tendency to flat-foot; no scoliosis; olecranon and *patellar reflexes normal.* Sometimes headaches, often vertigo; trunk ataxia; motor ataxia in using arms and legs; some nystagmus, which disappeared. Intelligence good.

Whatever this case is, it is not a typical case of Friedreich's ataxia. The history gives unusually copious and varied abnormalities of sensation, and as the author himself says, the origin from a blow on the head, and the cerebellar ataxia, are suggestive of tumour. The other symptoms all seem to point in the same direction (vomiting, polyuria, retention of knee-jerks, giddiness, &c.).

A case is reported by T. S. Wilson[39] (Case III.), as interesting from showing the symptom of glycosuria. The patient died at the age of 10 of diabetic coma, had been ill for five years, showed ataxia of gait, and slowness and hesitation in speech; a certain amount of clumsiness of hands; absence of knee-jerks; equino-varus; lateral curvature. There was no choreiform unsteadiness; no tremor on movement: great emaciation (from the diabetes, no doubt).

The case is certainly very interesting, and though the details are meagre, I should be more inclined to class it with the peripheral neurites than with Friedreich's ataxia. The sensibility was normal, but this may occur in a case of diabetic neuritis.

Case IV. of the same physician seems really to be what it was sent into hospital as, viz., St. Vitus' Dance. The unsteadiness became, in a fortnight from onset, so marked that the least thing would knock him down. There were muscular weakness; lordosis; increased knee-jerks; spastic tendency. Much improvement in hospital. Whole duration of illness to time of examination, only eight months.

Putting aside such cases as extremely doubtful, one finds considerable differences in those that remain.

Ladame's,[1] M'Caw's,[31] Baskett's,[76] Mackenzie's,[48] and Berdez's,[79] to name only a few, may be looked on as quite typical.

My Case IV. shows the essential points, namely, onset in childhood ; first symptom (ataxia) in legs ; progressive course involving legs and arms ; affection of speech ; swaying and choreiform jerks ; absence of knee-jerks ; Romberg's sign ; nystagmoid movements ; and amongst characteristic negative signs, absence of sensory defect ; clearness of intellect ; normal pupils ; normal ocular fundi.

On the other hand, there are some points of divergence. Though the disease is now far advanced, there is no club-foot or extension of great toe and no marked scoliosis. Cases reported by Dreschfeld,[50] D. Inglis,[20] Besold,[54] (Cases III. and IV.), Zabludowski,[84] and many others quite undoubted, show no abnormality in this respect.

The hesitancy in beginning the act of micturition is not very uncommon, and is present in Case II. The cramp, curiously enough, affects only the left leg. According to Griffith, spasmodic contraction of muscles occurred in 21 of his 143 grouped cases, and was usually in the lower extremities.

Chorea is one of the diseases for which Friedreich's ataxia has several times been mistaken—Ladame, Londe and Lagrange,[72] Bramwell,[12] first case, one of Ormerod's (*Med. Chir. Trans.*, vol. lxviii., p. 149), &c.

It is to be distinguished by the slowly progressing character, the absence of knee-jerks, and the character of the involuntary movements. In marked cases of either disease there can be no difficulty. The tongue, for instance, in a choreic is jerked up and down, in and out, while in an ataxic it shows an irregular tremor with no rapid jerks. The face is usually not affected in Friedreich's disease, though in Cases I. and II. slightly marked exceptions may be noted. Chorea does occur in families. " Though the proportion is not great, the family tendency in some cases is very marked." (Gowers.)

Huntingdon's chorea is hereditary, begins in adult life, is accompanied by mental symptoms, and is always fatal.

Hereditary Cerebellar Ataxia.—It is by no means certain, as yet, whether this is to be looked on as a definitely marked disease with independent pathology. So far it would appear that the symptoms may vary to an extraordinary extent, and the autopsies on which Marie[46] based his reasoning amount to only two amongst the sixteen cases that he reckoned up as belonging to the new type.

Londe wrote a lengthy thesis on the subject (*De l'hérédo-ataxie cérébelleuse*), and added three new cases.

Marie recognises in all the character of staggering and cerebellar inco-ordination, and connects this with the cerebellar atrophy found in the two autopsies. He is supported in this by the most recent observations on the function of the cerebellum.

Both Friedreich's disease and the new type are family diseases, the latter beginning more commonly in adult life, the former in childhood. But as we have seen, Friedreich's disease may begin at twenty-five, and on the other hand, Fraser's patient (*Glasgow Medical Journal,* 1880) showed signs in infancy. This is one of the two cases of cerebellar hereditary ataxia in which an autopsy was made.

The various other points, when gone into, show a close parallelism in the two types. The symptoms of hereditary cerebellar ataxia may be summarised thus from Londe:—

(1) Slow and gradual affection of legs, in standing or walking.

(2) Occasional pains in legs and loins.

(3) Usually in three years or so, uncertainty of hands and arms.

(4) About the same time speech and vision involved. (In Londe's own cases the eyes were not affected, if one excepts nystagmiform movements).

(5) Reflexes remain or are increased. This is of course very exceptional in Friedreich's disease.

(6) Sometimes mental enfeeblement.

(7) Death at advanced age, usually from intercurrent disease, frequently of the lung.

It is much easier to say that the cases collated by Marie and Londe are not cases of Friedreich's ataxia, than it is to admit that they should be put together to form a homogeneous group. But the theory put forward by Marie is at least plausible—that the same kind of degenerative process may be at work, involving the cerebellum only or mainly, in his cases—the spinal cord in Friedreich's disease.*

RELATION OF MENTAL DEFECT TO FRIEDREICH'S ATAXIA.

In cerebellar hereditary ataxia *mental enfeeblement* is often present. This leads one to consider the question "What weight should be attached to this point in making a diagnosis of Friedreich's ataxia?"

The pure type of this affection involves no mental change unless occasional irritability and a tendency to uncontrollable laughter; even this is exceptional. In fact, if we exclude the nystagmus and the speech affection, in regard to the causation of which we are quite in the dark, we have no evidence of involvement of the cerebrum, one might almost say, of the encephalon.

When the mind is affected we may take it for granted that the higher cerebral centres are diseased. We have seen that this condition is now and again present in cases that in other respects correspond to Friedreich's ataxia, but an examination of some cases recently reported makes one disposed to think that they should be regarded, not as Friedreich's ataxics, but as transitional to other types, *e.g.*, cerebral diplegia.

Amongst these I would reckon Nolan's[63] three cases of Friedreich's ataxia with congenital idiocy. These were a sister, aged 22, and two brothers, aged 15 and 10. Their mental condition was one of low intelligence; volition rarely manifested; memory defective; no delusions; speaking only in answer to queries; anxious to please; good humoured; asexual. All had nystagmus, internal strabismus; imperfect laboured articulation, many sounds not being uttered; ataxia in the arms and legs, with Romberg's sign.

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* Compare Dr. A. Meyer's paper in the last (Autumn) number of BRAIN, with a reading of which I have been favoured by the Editor.

The thyroid was enlarged in the two elder. "Universal partial analgesia," and "drop-ankle" were present in the same two. Knee-jerks absent in oldest and youngest, exaggerated and combined with ankle clonus in the other. No scoliosis or Friedreich's foot; a "suggestion" of *main en griffe* in two; no visceral disturbances, or ocular troubles other than those referable to the external muscles. The true jerky or choreic movements do not seem to have been very marked. The disease was observed soon after birth.

There are indications here of an extensive defect of development in the whole nervous system. The cases seem to be more closely allied to cerebral diplegia than to Friedreich's ataxia.

Nearly related to these are the cases of a brother and sister (Bouchaud[60]), who at the ages of six and seven began to show mental enfeeblement, inco-ordination of the four extremities, and tabeto-cerebellar gait. No paralysis, nystagmus, or kypho-scoliosis. Speech slow but well articulated. The dementia steadily increased. Sight became lost; speech unintelligible; chewing and swallowing slow; rigidity of muscles increased, though knee-jerks were in one case absent, in the other diminished. The autopsy showed double sclerosis of lateral pyramidal tracts, diminution of sheathed fibres in cortex, anterior and posterior columns healthy; ganglion cells of anterior and posterior horn diminished in number.

The same category may be said to include Vinaj's[89] family, about which he asks "Is this a hitherto unrecorded kind of case or a modification of Friedreich's ataxia?"

Father, mother, and four children alive, second dead. Signs of nervous predisposition.

(1) Eusebio, well till age of 5, then difficulty in speaking and walking; intelligence affected, ending in idiocy; steady degeneration. When 11, paralysis of legs; paresis of arms; physical degeneration; no control of bladder or bowel.

(2) Marietta showed same symptoms at 5; died of accident at 9; by that time had lost the power of speech.

(3) Iside, age 6; slight nystagmus; knee-jerks normal;

gait uncertain; staggering; speech hesitating; scanning; memory weak.

(4) Egidio, aged 5, large head; unequal pupils; nystagmus; hesitating speech, pronunciation like a child of three; normal jerks.

(5) Healthy child of fifteen months.

In these cases the evidence against a diagnosis of Friedreich's ataxia consists in retention of knee-jerks, absence of talipes and scoliosis, and, most important of all, early and marked loss of intelligence. The family tendency, age of onset, ataxia, affection of speech, nystagmus and preservation of sensibility bring it into relation with that disease. The author rejects as diagnoses:

(1) Little's disease (origin; absence of special gait; absence of spastic rigidity; psychic phenomena).

(2) Spastic tabes.

(3) Primary progressive myopathy.

(4) Chronic encephalitis: primary cerebral sclerosis of infancy (time of onset; normal mode of birth; absence of convulsions, of contractures, of paresis or paralysis; the mental phenomena).

The cases are quite analogous to a number of others described by various authors, many of which are brought together by Higier[78] in his study of the rarer forms of paralysis of a hereditary and family character. After detailing four cases of his own in one family, he reviews the cases of cerebral diplegia reported by Sachs, Freud, Strümpell, Erb, Tooth, &c. The age of onset and the character of the affection are pretty constant in all, but each family has its distinctive characters. If one takes as the chief symptoms, onset in early childhood, spastic or flaccid paraplegia, idiocy, nystagmus, affection of speech and sight, one sees that Vinaj's and Nolan's cases are perhaps more nearly related to this group than to Friedreich's ataxia.

Higier's own cases are specially interesting, in relation to the point now under discussion, from the fact that they showed disturbances of co-ordination; weakness and awkwardness in upper extremities; intention-tremor; great difficulty in sewing, knitting, and writing; in lower extremities weakness and stiffness followed by spastic paresis in gait, in one spastic ataxia for a time. He draws attention to the "little-known fact" that inco-ordination does now and then occur with increased tendon reflexes, quoting reports by Sachs, Haushalter, König, Dreschfeld. I may abridge the descriptions of Haushalter's cases:—Parents near blood relations, father alcoholic. Four brothers and sisters, normal births. Two eldest affected, now 11 and 10 years old. Symptoms first while at breast; rapid advance; inco-ordination of four extremities; fixed position of arms, of hands and fingers; slight rigidity in extremities, more in tongue and face; choreiform jerks when at rest; increased knee-jerks; *pes equinus;* anomalies of speech; disturbances of swallowing; imbecility. Nystagmus only in eldest.

These cases differ from Friedreich's ataxia in the retention of knee-jerks, the imbecility, the rapid advance at an early age and some other minor points, but they are transition forms between this disease and cerebral diplegia.

Collins[73] reports a case quite allied to those transition forms:—A child, 11 years old, of tubercular heredity (whose brother died in his second year, defective mentally and nystagmic), began to walk at end of first year. From third year gait stiffer, often falling; high myopia in fourth year; did not get on at school; when seen at age of 11, choreic movements of lower extremities; speech indistinct; exaggerated action of mimetic muscles under emotion; old face; gait cerebellar-ataxic, legs kept apart; ataxia of arms, especially left; increased knee-jerks; occasional ankle clonus; explosive ataxic speech; imbecile; sensibility and sphincters intact; no muscular atrophy; progressive paresis; slight nystagmiform movements; slight club-foot.

Here there is clearly a defective developmental condition of the nervous system in both cord and brain.

It is interesting to note how often in these cases which present a combination of the symptoms of Friedreich's ataxia with idiocy, the knee-jerks are retained. This tends to confirm the very high position of Westphal's sign as a diagnostic mark.

To the above list I might add the two cases of Londe and Lagrange,[72] who showed imbecility with the exceptional athetoid attitude in walking, and Hoffmann's[71] case, where there were imbecility and epilepsy, a case he himself records as doubtful in regard to diagnosis.

One sees from this review that when the degenerative process has involved not only the spinal cord, but the higher cerebral functions, other symptoms besides simple imbecility or dementia are almost invariably added to the symptoms of Friedreich's ataxia. And in such cases there must always be a doubt as to how far the changes in the cord, with their accompanying symptoms, are secondary to the cerebral affection, a doubt which a *post-mortem* examination might not clear away. Mental defect seems a much more important sign than the retention of the knee-jerk in making one doubt a diagnosis of Friedreich's ataxia.

Disseminated Sclerosis.

We pass now to consider the differentiation of Friedreich's ataxia from disseminated sclerosis. This disease is said never to occur in families (Marie), but a difficulty may arise when one meets an isolated case of Friedreich's ataxia. A good number of cases have been recorded of its occurrence in early childhood, but the proof of this anatomically was first given by Eichhorst in a paper "über infantile und hereditäre multiple Sklerose" (*Arch. für Path. Anat. und Physiol.* &c., Bd. 146, Hft. 2, p. 173). A well-marked case of insular sclerosis can present no difficulties; the intention tremor is coarse and jerky, much more so than in Friedreich's disease; the nystagmus is more pronounced; the course of the disease is liable to fluctuations in much greater degree; the tendon reflexes are exaggerated; ocular paralyses and affections of the optic nerve are frequent.

But the group of symptoms due to insular sclerosis varies much. The gait is at times cerebellar in type; knee-jerks are occasionally absent; scoliosis may be present; the speech may be similar to that in Friedreich's disease.

Brissaud (quoted in Londe's thesis) says it may be absolutely impossible to distinguish an isolated case of *hérédo-ataxie cérebélleuse* from a case of *sclérose en plaques*, and the following cases may show the difficulty in diagnosing it from Friedreich's disease. Bouchaud[66] describes this one as " Maladie de Friedreich " :

Boy, aged 18, always rather delicate, fairly sharp at school. First symptoms at 12, viz., awkwardness of hands; tremors of upper extremities; difficulty in walking. Apprenticed to saddler at 14, but made no progress. On examination asymmetry of face (left smaller); left arm and leg more awkward and weak than right; intelligence much below normal; speech difficult to understand; does not speak much; no choreiform instability when at rest; static ataxia when trying to stand; inco-ordination; intention tremors; gait not tabetic; staggering, requires support; " tendency " to talipes equinus; exaggerated extension of great toe; knee-jerks both retained, left exaggerated; nystagmus marked; slight lateral curvature; acuteness of vision much diminished; hallucinations; no evidence of loss of sensibility.

This case differs from Friedreich's disease in quite a number of particulars, especially the mental condition, retention of knee-jerks, affection of vision, gait, asymmetrical appearance of face, greater awkwardness on one side. On all these points a diagnosis of disseminated sclerosis would be much more adequate.

Ewart[11] gives a case of a female, aged 19, of "isolated" type. Symptoms began at 16. Menstruation first irregular, then ceased; slight affection of speech, only temporary, a year ago; knee-jerks exaggerated; slight tremor of hands on purposive movements; slight lateral nystagmus; marked ataxia of lower limbs; slight loss of sensation; functions of brain intact.

He thought disseminated sclerosis could be excluded by the insufficient degree of tremors and nystagmus and the normal cerebral condition. Friedreich's disease is, however, a doubtful diagnosis, on account of the disappearance of speech affection and the exaggeration of the jerks. These points would bring it nearer to disseminated sclerosis.

Probably, in a case like this, continued observation of the course would settle the question of diagnosis.

Locomotor Ataxia.

The diagnosis from ordinary locomotor ataxia can present little difficulty. A comparative table of the symptoms is given in Ladame's paper. A difficulty comes up when cases of Friedreich's disease have to be distinguished from tabes commencing in childhood. In the latter there is usually a syphilitic heredity, but not in the former. In a summary of ten cases Hildebrandt of Berlin (27) found that six began before tenth year, four before fourteenth. In some cases ataxia of extremities takes the leading place, lancinating pains in two, girdle sensation in four. Loss of patellar reflexes in all cases, eyes three times normal, nystagmus twice present, reflex paralysis of pupil twice, disturbances of sensibility in only half, disturbances of bladder six times. Moreover, the speech often shows disturbances on account of slight cerebral affection. Fortunately the symptoms are so grouped usually that there is not much difficulty in reaching a decision.

Raymond[91] discusses this subject at some length in a recent paper. He first records two cases, father and son. The father was a well-marked tabetic, showing, in addition to pre-ataxic symptoms, disturbance of superficial and deep sensibility, loss of knee-jerk, urinary incontinence, ataxia of the four limbs, characteristic gait, Romberg's sign, and marked eye affections. Onset in 1883, a few years after his marriage. Of his family, two girls and one boy were healthy, but the youngest boy had a nervous disease. He had always been rather weak in mind. In October, 1895, at the age of 15 he noticed one evening that he could not run, though he could walk. There was loss of muscular power; he soon found difficulty in climbing a ladder; he let things fall, and lost sensation of touch in his hands. He had no pains, but a sensation of wearing tight boots, even when his feet were bare. Gradually he lost confidence in movements, his gait became uneven, and his speech was embarrassed. In

January, 1896, this was his state: Development good; slight upper dorsal scoliosis; convex to right; tabeto-cerebellar gait; Romberg's sign; much impaired sense of touch in hands; impaired sense of posture; loss of deep reflexes; fibrillar and jerky tremors of tongue; nystagmiform jerks; frequent foolish laughter; intellectual functions limited. At present (September, 1897) condition rather worse; sight gone; (discs "tabetic"); jerky movements of limbs and trunk more pronounced, especially on effort.

In discussing this case, Raymond points out certain resemblances to Friedreich's ataxia, viz., gait, scoliosis, speech affection, nystagmus, jerkings, loss of knee-jerks. On the other hand, it differs in the marked affections of sensibility of skin and joints, and the optic atrophy. The case resembles tabes in these respects, in the loss of jerks and in the motor inco-ordination, but differs in the absence of a pre-ataxic stage with lightning pains, Argyll-Robertson phenomenon, urinary troubles; also in the swaying character of the gait, the nystagmus, the scoliosis and the speech affection. It is, in short, a hybrid case, a transition form between tabes and Friedreich's ataxia. Going into the question of juvenile tabes, he points out its great rarity; he himself has not met with a case in five hundred of locomotor ataxia. Only ten cases can he find in literature, with a doubtful eleventh, but these are all very characteristic, not differing from the adult form unless in a greater tendency to early development of optic atrophy, this being unaccompanied by any delay in the advance of the malady. Most of the cases published as juvenile tabes are cases of Friedreich's ataxia, or closely allied thereto, and amongst those Raymond would place two recorded by Freyer, and quoted by Hildebrandt in the paper above referred to.

But tabes in the adult may take abnormal forms. Auscher's[30] "case of Friedreich's ataxia," with autopsy, presents several points of close correspondence to locomotor ataxia. The onset was not till the twenty-fifth year; there was mental degeneracy, and abolition of light reflex. The patient was a prostitute, and there were no other members of her family affected. She showed marked ataxia of limbs;

choreiform movements of head and trunk; double equinovarus; loss of knee-jerks; nystagmus and affection of speech. Sensibility was retained, though in a case of marked mental defect it must be difficult to test it completely. Some lightning pains were present. The autopsy may be summarised thus:—Meninges normal; cord much diminished in size, like that of a child seven years old; cerebrum and cerebellum much smaller than normal, but free from microscopic changes; *posterior horns* much atrophied in whole length, worst in dorsal region; atrophy of fibres of Clarke's columns, number of cells in them diminished; *posterior columns* sclerosed in whole length, worst in lower part, Goll's columns being more changed than Burdach's; only a few medullated fibres in Goll's columns in a network of fine neuroglia fibres; *Lissauer's zone* normal in lumbar and cervical portions, perhaps slightly degenerated in dorsal; posterior roots and antero-lateral columns normal; motor nerves normal; sensory nerves showed a great number of non-medullated fibres, or fibres with an extremely thin sheath, perhaps embryonal.

The sclerosis showed what Déjerine had a few months before described as a pure gliosis: great abundance of fine neuroglial fibres arranged in whorls, absence of granule cells, no thickening of vessel walls or of septa connected with pia mater.

From the clinical account, as well as from the autopsy, one would prefer a diagnosis of locomotor ataxia, with an ill-developed brain, the congenital mental defect being possibly aggravated by a progressive paralysis, as Schultze suggests.[58] Criticism of a case like this is most desirable, for from it the following conclusions have been drawn:—

(1) Friedreich's disease may start as late as 25.

(2) Cerebellar defect may account for the clinical signs of Friedreich's ataxia (Senator).

(3) Mental degeneracy, abolition of light reflex and other exceptional conditions may occur in Friedreich's disease.

(4) The motor nerves may be normal, and the sensory show certain changes in Friedreich's disease (Leyden and Goldscheider).

(5) A full confirmation of Déjerine's views on the peculiar character of the histology in Friedreich's disease.

In making such inferences from a single case, the doubtful nature of the diagnosis must be borne in mind.

Disease of Cerebellum may cause symptoms like those in Friedreich's disease: reeling gait with a zig-zag character, difficulty in standing, nystagmus, loss of knee-jerk (usually not constant); but there is no tabetic character in the gait; the choreiform movements are probably absent; "the arms are usually steady, rarely they present some jerky inco-ordination" (Gowers), and in the case of tumour, pressure symptoms are generally present. Some cases of simple atrophy or hypoplasia of cerebellum have been described, but the symptoms are variable. In König's[65] case there was congenital cerebral diparesis, but no affection of inco-ordination at all, although the hypoplasia was very well marked.

"Oppenheim has observed cases of *cerebellar sclerosis*, and has had one anatomically investigated by Arndt. He regards cerebellar sclerosis as associated with a clinical picture composed of vertigo, cerebellar gait, ataxiform disturbances of movement, weakness of bladder, difficulty of articulation and motor weakness of the extremities" (Leyden and Goldscheider, *loc. cit.*, p. 597).

Although there is undoubtedly a general resemblance here, to cases of Friedreich's ataxia, there would probably not be much difficulty in distinguishing actual cases.

Cardossi[19] publishes a case of "*a rudimentary form of Friedreich's disease*," which he admits is by no means typical.

Giuseppe P., aged 40, has a son aged 18, who has had tremor in the right hand from the age of 13. He himself began to have tremor in writing at 10. Right arm and body affected; never so bad as to prevent him from attending to his usual occupation; gait, tabeto-cerebellar; has had phthisis for last ten years, not much advanced; oscillation of body when standing, brief, rhythmical, not well marked; intention-tremor in upper limbs, as in disseminated sclerosis; slight tremors of tongue; speech slow and hesitating; no nystagmus; pupils normal; sensory system normal; knee-jerks much diminished; no paresis or paralysis; no deformities of feet.

The symptoms are of too mild a type and too few for Friedreich's disease of thirty years' duration, and they can be resolved into tremor, but apparently not that of chronic chorea, nor of paralysis agitans. Possibly a functional disorder allied to one or other of those.

In this discussion on *diagnosis*, I have not attempted to set forth exhaustively the differences between Friedreich's disease and the various affections likely to give rise to difficulty. My desire has been rather, by giving groups of cases from recent medical literature, to bring out the close relationships that bind one type to another. From a text-book stand-point this would be most confusing, but one cannot be too often reminded of the somewhat arbitrary character of the boundaries. The more numerous the doubtful cases that are published the better, though unfortunately there is sometimes a tendency to magnify this symptom or minimise that, so as to get the case included in a favourite type.

PATHOLOGICAL ANATOMY.

The following are the more important records of *post-mortem* examinations in Friedreich's disease:—

(1-5) Friedreich and Schultze. (6) Everett Smith, *Boston Med. and Surg. J.*, 1885, vol. cxiii. (7) Newton Pitt, *Guy's Hosp. Rep.*, vol. xliv. (8-9) Rütimeyer, *Virch. Arch.*, vol. cx. (10) Letulle and Vaquez.[3] (11) Blocq and Marinesco.[5a] (12) Déjerine and Letulle.[2 and 6] (13) Guizetti.[49] (14) Mirto.[37] (15) Michell Clarke.[57] (16) Burr.[74] (17) Dana.[77]

Autopsies by Menzel, Brousse, Kahler and Pick, Erlicki and Rybalkin, Rossi and Auscher, are rejected from the list as dealing with cases that can hardly be reckoned examples of Friedreich's ataxia.

For adequate reasons see Ladame,[1] Tedeschi,[85] and Schultze.[58]

Most of the cases that have been fully described have shown extensive system lesions. They have been summarised by Ladame in 1889, and more recently Tedeschi has given an analysis of 15 up to 1896. The most common conditions are diminution in bulk of the cord, sclerosis of posterior columns (especially Goll's), and degeneration of Clarke's columns. Next in frequency is degeneration of the direct cerebellar tract, and the antero-lateral ascending tract of Gowers. The crossed pyramidal tract may be partially involved, but it is thought that the true motor fibres are unaffected unless in very advanced cases. In one of Friedreich's cases the direct pyramidal tracts were involved. Apparent involvement of motor tracts is explained by the presence in them to a greater or less degree of neurons with other than motor functions. The "system" arrangement of fibres is, in short, not an absolute one. Occasionally changes in the spinal ganglia have been observed. The posterior roots have sometimes been found atrophied. Changes in form of the central canal, and sometimes peri-ependymal sclerosis, have been seen (Déjerine and Letulle).

Ladame's opinion that the zone of Lissauer (fine fibres at outer side of posterior column stretching from apex of posterior horn to periphery) always remains intact can no longer be supported. He looked on this as a point of distinction from tabes. But in several cases (Letulle and Vaquez, Blocq and Marinesco, Guizzetti, Mirto) it has been found more or less extensively altered.

The degeneration of Clarke's column, especially of its ganglionic cells, seems associated with the sclerosis of the direct cerebellar tract. The view that these cells are trophic centres for the tract fibres is thus confirmed. In early tabes Clarke's columns are affected. There is an absence of the fine fibrous network, while the coarse fibres, the ganglion cells, and the fibres of the direct cerebellar tract arising from them are unchanged. In advanced tabes Clarke's columns are almost entirely degenerated, and then the cerebellar tracts are said to be sclerosed (*see* Leyden and Goldscheider,[87] p. 509).

There appears to be no constant anatomical character which would allow one to say from examination of a section of the cord that one had to do with Friedreich's disease, and not with tabes, or a combined sclerosis. Déjerine and Letulle have endeavoured to find a distinctive character in the nature of the sclerosis. Those authors believe that the sclerosis in the posterior columns is purely neuroglial, a gliosis, consisting of fibrillæ intercrossed in different directions, and forming whorls like those described by Chaslin in the brains of epileptics. Further, the pial prolongations into these columns are unaltered, and not more evident than in a healthy cord. In tabes, on the other hand, there is thickening of the septa of the pia mater, and of the prolongations which start from them, thickening of the neuroglial tissue between the nerve tubes, very pronounced changes in the vessels, &c. A vascular sclerosis such as this they found in the *lateral columns* in their cases.

To meet the possible objection that they are making an extensive generalisation from two cases, they point out that similar changes have been seen by several authors, but that they have not interpreted them in their true sense. They quote to this effect from the histological descriptions of Friedreich himself, Newton Pitt, Rütimeyer, and Blocq and Marinesco. Auscher, in his doubtful case, showed a similar condition at the Société de Biologie, July 26, 1890; and Déjerine, after examining his specimens, pronounced them examples of pure neuroglial sclerosis.

Schultze, of Bonn, re-examined by recent methods the cord of a case observed and described by Friedreich and himself in 1881, and he failed to find any justification for the distinction in the nature of sclerosis thus defined by Déjerine and Letulle. The whorls of fibrils were not present. The vascular thickenings were found in the posterior columns, especially between the columns of Burdach and Goll. He sums up his re-examination thus: No proper chronic meningitis. The degenerations are essentially a degeneration of the posterior roots and their connections in the posterior columns and posterior horns, disease of Clarke's columns, of the cerebellar fibres, and the pyramidal tracts—this so far as concerns the spinal cord.

Michell Clarke[57] describes the autopsy on a patient aged 14, a typical case except that the knee-jerks were normal, and that during the last few weeks of life he showed symptoms of cerebral tumour (cerebral vomiting, intense headache, optic neuritis, coma, &c.). A round-celled sarcoma was found growing from the under surface of the right half of the cerebellum, pressing on the pons and medulla. Apart from this no change in cerebellum. In cord no naked eye change except diminution in size. "No complete degeneration in any section, but a partial change chiefly in posterior columns, next most marked in lateral columns with involvement especially of the margin of the cord, with thickening in the pia mater and the walls of the vessels, and probably with some excess of the neuroglia."

A brother and sister were affected with the same disease. Had it not been for this, in view of the presence of a tumour in the cerebellum, one would have been apt to ask could not this have accounted for the symptoms. Certainly the symptoms were very severe at the end, and the tumour was apparently growing rapidly then, but it might have taken on an active character after a long comparatively latent course. The family had a strongly neuropathic tendency, and it is of course quite possible that two members of such a family should have Friedreich's disease and a third a cerebellar tumour without Friedreich's disease. Clarke himself states that the changes in the cord were by no means so marked as he was led to expect from the symptoms.

Dana's[77] autopsy is briefly recorded: it is accompanied by some photographs of cord-sections. In addition to smallness of cord, thickening of pia, sclerosis of posterior columns, sclerosis of lateral columns (crossed pyramidal and direct cerebellar tracts) and marginal sclerosis, he observed vacuolisation in the gray and white matter all through.

There is no note of the condition of Clarke's columns or Lissauer's tracts, nor could the author say whether the sclerosis was really a gliosis.

Burr's[74] autopsy was on a case that was eighteen years ill. He found (1) marked degeneration of Goll's columns, slighter of Burdach's, from top of cervical to lower lumbar cord; (2) degeneration of lateral and anterior pyramidal tracts; (3) degeneration of direct cerebellar tracts. The sclerosis of lateral columns was not systematic. (4) posterior roots much degenerated, anterior intact; (5) slight affection of posterior horns with degeneration of ganglion cells, while in anterior horns only a very few cells were atrophied; (6) degeneration of cells in Clarke's columns; (7) walls of vessels slightly thickened.

The following are the chief points in Guizzetti's [49] case, who died of heart disease at the age of 28, after the nervous affection had lasted eighteen years: small size of posterior roots, spinal ganglia and cord.

(1) Lumbar region—sclerosis of posterior columns except a portion immediately behind posterior commissure and inner border of posterior horn, and also Westphal's posterior limiting zone; numerous degenerated fibres in pyramidal tracts.

(2) Dorsal region—posterior columns (with exception of part as in lumbar region), pyramidal tracts (numerous fibres), Clarke's columns and Lissauer's band all involved.

(3) Cervical region—Goll's columns as seriously involved as lower down, Burdach's less so, more healthy fibres in pyramidal tracts. Longitudinal fibres degenerated even into the nuclei of Goll's and Burdach's columns in the bulb, the horizontal fibres remaining unaffected. Some altered fibres even in anterior pyramids of bulb, contrary to usual condition.

Posterior horns atrophic. Microscopic condition like that described by Déjerine and Letulle.

In peripheral nerves, diminution of coarse nerve fibres and presence of numerous fine fibres.

Mirto's [37] case (death after five years' duration of illness, at age of 19) had systemic degeneration of posterior columns (Goll's, Burdach's and Lissauer's) of lateral cerebellar tracts, of Gowers' tracts; slight degeneration in crossed pyramidal tracts disappearing in cervical region, advanced sclerosis of Clarke's columns; rarefaction of reticulum nervosum in anterior and posterior gray columns with partial cell-atrophy, partial degenerative neuritis in motor nerves (sensory not examined)—spinal ganglia affected as regards both cells and reticulum.

The latter two abstracts are taken from Tedeschi's critical digest, and I have not seen the clinical reports of these cases. But, supposing them to be typical, nothing of much importance has been added to what was known in 1889 to Ladame, or indeed Friedreich himself. The posterior roots and ganglia have shown marked changes, and in this respect and the coincident or consecutive affection of the posterior columns the similarity to tabes is obvious. In two cases (Guizzetti's and Mirto's) the principal nerves showed embryonic characters. In Mirto's the sensory were not examined, but the motor nerves were affected. Auscher's doubtful case presented similar appearances in the sensory nerves.

PATHOLOGY.

Various theories have been suggested to establish some causal relationship between the anatomical basis and the clinical symptoms. The general assertion that in a case of typical Friedreich's ataxia the symptoms are mainly spinal is one which has been widely though not universally accepted. As for the nystagmus, the affection of articulation, the giddiness and certain choreiform movements, though their origin is in all likelihood intra-cranial they have as yet no morbid anatomy. The strongest critic of the spinal origin of most of the symptoms is Senator.[34] [35] He has maintained the view that the disease is essentially *cerebellar*, that in its most outstanding features it depends on an arrest of development of the cerebellum or of single physiologically important parts of it, and that it is only in a somewhat advanced stage that other symptoms appear, referable to super-added degenerations, in cord and cerebrum. The cord changes may be concomitant or consecutive. In confirmation he quotes Marchi's conclusion from experiments: "Destruction of cerebellum entails degeneration of lateral portions of antero-lateral columns, of anterior part of direct cerebellar tracts and of a certain number of fibres in the pyramidal bundles." He says that we know nothing of the symptoms associated with disease of Goll's columns alone, that a separate or simultaneous affection of Burdach's would give a clinical picture like that of tabes dorsalis, and would be more likely to do so as certain other peculiar changes in tabes (affection of Clarke's columns, of Lissauer's zone, atrophy of posterior roots and degeneration of peripheral nerves) are not wanting in hereditary ataxia. Moreover, by reference to the autopsies he finds that the condition of the cerebellum was not mentioned in eight out of the fifteen he analysed. In three cases of the other seven there was a recognised atrophy (Menzel's, Rossi's, Auscher's). In three cases nothing wrong was seen microscopically, and in one nothing either with naked eye or microscope. This is the weakest point about the theory—the want of anatomical evidence—and this objection has not been removed by the later *post-mortem* examinations, though Clarke's case given above is interesting in this relation. As regards the three cases he quotes, we have seen how unusual in some important respects Auscher's case was; as for Menzel's, it is generally regarded as not a case of Friedreich's disease, and Rossi's also seems to deviate considerably from the classical type.

The small size of the spinal cord in Friedreich's disease seems to have struck all observers, and Friedreich and Schultze first advanced the view that the columnar degenerations were secondary to an *arrest of development in cord and medulla*. Shrinking of the cord no doubt occurs subsequent to the degeneration as the connective tissue takes up less bulk than the nerve tubes, but it is questionable if this would account for all the difference; it would hardly explain why the cord is so much less in this disease than in other scleroses, even if the points emphasised by Schultze are allowed their full weight. This author speaks of the general atrophy of the cord in tabes in the dorsal and lumbar regions as due to long disuse of the lower extremities. So in Friedreich's ataxia one would, he says, still more look for such atrophy, on account of the inactivity enforced in childhood and the longer duration of the disease than in the case of locomotor ataxia.

The theory of developmental error is supported by other facts—the presence of numerous fibres without sheaths in peripheral nerves (Guizzetti, Mirto), anomalous development of central canal of cord (Déjerine, who quotes similar observations by Ormerod, Pitt, Everett Smith); condition of syringo-myelia (Friedreich's fourth case, quoted by Schultze).

No satisfactory explanation has yet been given as to why the cord should be able to perform its functions for a series of years varying from two to twenty or more, and then show signs of inadequacy. The demands made on the spinal cord of a young lad are great enough to test its capabilities most thoroughly. In certain families, and certain isolated cases, parts of the central nervous system seem to be badly constructed. They may be looked upon as unfit to bear a long strain, and their decay and death gradually come about; not that the change is analogous to a senile change; at least the differences are so great as almost to hide any analogy that may be supposed to exist. If the process of disease or decay has once been started, it is likely to be aggravated by the struggle for existence among the cells that is taking place constantly at all ages, and so actively in youth. The neuroglia rapidly increases, and not only fills the space formed by shrinking of the nerve-tubes, but compresses and destroys healthy nerve tissues (Roux and Weigert, quoted by Edinger).

Edinger[90] a few years ago brought forward his *repair* theory, to explain the condition found in several nerve affections. In any exertion there is waste of nerve-cell and nerve-fibre, compensated in health by repair. If nutrition is defective through any cause, as a toxin (syphilis, diphtheria), or injury, or hereditary nervous weakness, repair will be insufficient and degeneration may be the result. Some of the cord tracts seem more easily acted on than others, viz., the posterior columns, pyramidal tracts, and, less frequently, the cerebellar tracts. A greater strain may be put on certain nerve-lines than on others. Thus, taking up tabes, he would explain the involvement of the nerve apparatus connected with the erect posture, the anæsthesia of soles (pressure on soles), affection of muscle sense, of eyes, &c.

In Friedreich's ataxia he thinks the progress of the disease may be explained as the legs and arms are used more and more with the progress of years. There is some abnormal weakness, either congenital or precipitated by an acute disease. The posterior and lateral tracts which are most used thus become affected. Here, however, Edinger admits there are serious objections to the adequacy of his theory. Why are other parts of the nervous system exempted through life? Why do the pupils, the bladder and the brain not show more disturbance than they do? No answer is attempted, but the very elementary nature of our knowledge of the physiology of certain parts of the nervous system is emphasised.

Dr. C. Macalister (*British Medical Journal*, April 8, 1893) suggests that the tendency to degenerate in certain diseases, such as pseudo-hypertrophic paralysis, may be due to the diminution in the functional activity of some gland whose secretion exercises a controlling influence over the nerve elements. Stawell[67] would extend the theory to cover cases of Friedreich's ataxia.

It will be evident that the above theories, however suggestive, do not by any means suffice to explain the whole disease in any large and general way. This symptom or that may be elucidated through the aid of other affections, perhaps more thoroughly understood. Atrophy of muscles, for instance, as of the interossei, may be referred to degenerations of their trophic cells in the anterior horns. Atrophies in this disease are not so extensive or frequent as to lead one to expect any very striking evidence of wasting of ganglion cells, but such wasting has been discovered.

The *ataxia* seems to be different from that of tabes, both clinically and pathologically. The cerebellar character may be referred, if not to the cerebellum, at least to the tracts conveying impulses to the cerebellum. The direct cerebellar and the antero-lateral ascending tracts are longitudinal commissures between successive spinal segments and some part of the brain (Foster, "Physiology," par. 580). Another suggestion is that the posterior columns are the main tracts for conveying motor impulses downwards (Inglis, *Journal of Nervous and Mental Diseases*, January, 1892). Hughlings Jackson's view that cerebellar ataxia is due to weakness of the back muscles, and to the consequent attempt of the legs to correct the weakness of the back, is strengthened by such cases as those we have been considering. (For valuable discussion of this subject see paper by Ormerod, *British Medical Journal*, 1894, vol. ii., p. 1291.)

But the ataxia is not only cerebellar, it is tabetic also. The muscle-sense to whose impairment the most widely accepted theory refers the ataxia of tabes, is usually, though as we have seen not invariably, well preserved in Friedreich's disease. With a pathology so alike, as regards the posterior columns, one is tempted to think that whatever explanation is adequate for the ataxia and absence of jerks in tabes is likely to be sufficient *pro tanto* in the case of Friedreich's disease.

THERAPEUSIS.

Almost all writers on this part of the subject speak in the most hopeless tone. However, Zabludowski in his patient, a little girl of 9, obtained decided improvement by a four weeks' course of massage. The case was an isolated one, and was exceptional in some minor points; for instance, regurgitation of fluids through the nose occurred in swallowing, formerly solids also had been thus returned. The "cure" so improved her that she seldom fell, walked straight, and could feed herself without letting the spoon drop. Massage increases the functional capacity not only of healthy muscles but of those whose activity is impaired by direct or indirect causes. The nerves are better nourished through the improved circulation. Nerves and muscles only partially diseased may take up the work of those that are further gone. To this one might add Weigert's conception of the mode of action of a stimulus. "Neither cell nor organ is directly strengthened by the stimulus; the apparent strengthening is due to the fact that in action portions are used up and replaced by new fresh portions, a substitution which in many cases occurs in excess." (Edinger.[90]) Improvement thus obtained can hardly be anything but temporary. Case IV. had a month's massage in hospital but without benefit. It is clear, however, that rest is the worst thing possible for such cases; they should be encouraged to take exercise, and to go through such actions as will best bring their co-ordinating faculty into use. Touching definite points, picking up small objects and placing them on marked spots, writing, walking without or with aid—these and other exercises employed in the mechano-therapeusis of tabes should be useful in Friedreich's ataxia.

LITERATURE.

(1) LADAME, "Friedreich's Disease." BRAIN, vol. xiii., p. 467, 1890, a critical digest, with full bibliography to 1889.

(2) DÉJERINE ET LETULLE. "Etudes sur la maladie de Friedreich; sclérose nevroglique pure des cordons postérieurs." *La Méd. Moderne*, vol. i. No. 17. 1890.

(3) LETULLE ET VAQUEZ. "Un cas de maladie de Friedreich avec autopsie." *Comptes rend. de la Soc. de Biol*, p. 21, 1890.

(4) DÉJERINE. "Sur une forme particulière de mal. de Friedreich avec atrophie musculaire et troubles de la sensibilité." *La Méd. Moderne*, 1890. No. 25. Two excellent cases with paralysis of hands and feet.

(5) AUSCHER. "Anatomie path. de la mal. de Friedreich." *Sem. Medicale*, No. 32, July 30, 1890 (with remarks at the Société de Biologie, by Déjerine).

(5A) BLOCQ ET MARINESCO. "Sur l'anatomie pathologique de la maladie de Friedreich." *Archives de Neurologie*, pp. 118 and 331, 1890. Case of a girl, ill from age of 9, died at 19.

(6) DÉJERINE ET LETULLE. "Sur la nature de la Sclérose dans la mal. de Friedreich." *Semaine Méd.*, March 12, 1890.

(7) ROOK. "Four cases of Friedreich's disease." *Journal of Nerv. and Ment. Dis.*, p. 173, 1890. Claw-like hands in two. Autopsy on one, by Dana, 1896; *see* below.

(8) BROWN, C. H., of New York. Two cases, not related. *Journal of Nerv. and Ment. Dis.*, 1890 (abstract in *Neurol. Centralblatt*, 1892, p. 209). Both cases exceptional, doubtful.

(9) Erb. "Two cases of Friedreich's ataxia." *Deutsche Med. Woch.*, No. 46. 1893. Short account, knee-jerks.

(10) Mendel. "Friedreich's ataxia." Two brothers and a male cousin. *Deut. Med. Woch.*, No. 47, 1890.

(11) Ewart. *Lancet*, vol. i., p. 991, 1891. "Case of Friedreich's disease, with increased knee-jerk."

(12) Bramwell. *Atlas*, vol. i., p. 28, 1891-2. Case of Friedreich's disease, with essay on whole subject.

(13) Bramwell. *Atlas*, vol. i., p. 45, 1891-2. "Three cases of Friedreich's ataxia." Subsequent history up to death of two of those given by Herbert Bramwell, in *Brit. Med. Journ.*, October 2, 1897. Also a note on the histological characters in the cord of one of them, by Byrom Bramwell. The main interest of this is a confirmation of Déjerine's description of the wheel-like arrangement of the neuroglial fibres in the posterior columns. Otherwise the appearances were such as are commonly found.

(14) Bramwell. *Atlas*, vol. i., p. 178, 1891-2. "Notes of three new cases of Friedreich's ataxia."

(15) Lyman. "Juvenile or hereditary ataxia." *Intern. Clinics*, 1891 (knee-jerks increased).

(16) Baumel. "Considérations cliniques sur la maladie de Friedreich." *Nouv. Mercredi Méd.*, vol. ii., p. 49, 1891.

(17) Dana. *Keating's Cyclop. of Dis. of Children*, vol. iv., pp. 716-725, 1891. Article, with one doubtful case.

(18) Friedenreich. "Et tilfalde af hereditär ataxi. Friedreich's sygdom." *Hosp. Tid.*, 1891 (*Neurol. Centralblatt*, 1892, p. 211). One case, second in family, from birth, never steady on legs. Optic atrophy. Intelligence defective.

(19) Destrée. "Un cas de maladie de Friedreich." *Journal de Méd. de Bruxelles*, Nov. 5, 1891. One case with fulgurant pains, strabismus, and slight left ptosis.

(20) Inglis. "Friedreich's ataxia, its relation to conducting paths in cord." *Jour. of Nerv. and Ment. Dis.*, vol. xix., p. 30, 1892. An isolated case, no nystagmus.

(21) Sanger Brown. Brain, pp. 250-282, 1892. Twenty-one cases in five generations of a hereditary ataxia, not Friedreich's; cerebellar gait, increased jerks, visual troubles, affection of deglutition, late onset.

(22) Bramwell. *Atlas*, vol. ii., p. 22, 1892. "Two cases of Friedreich's disease in which the knee-jerks were not lost."

(23) Bramwell. *Atlas*, vol. ii., p. 70, 1892. "Three cases of Friedreich's disease with *main en griffe*."

(24) Hallion. "Des déviations vertebrales nevropathiques." *Nouv. Iconog. de la Salpetrière*, 1892.

(25) Leegaard. "Friedreich's sygdom (hereditär ataxi)." *Norsk. mag. f. Lagevidensk*, 1892, s. 444 (ref. *Neurol. Centr.*, p. 618, 1893). Three cases, acute onset, with fever and pains in limbs; spastic symptoms, with pains in limbs supervening on the ataxia.

(26) Marie. "Leçons sur les maladies de la moelle," 1892.

(27) Hildebrandt. "Uber tabes dors. in den Kinderjahren." Berlin, 1892 (abstr. in *Neurol. Centr.*).

(28) Geigel. "Uber Friedreich's hered. ataxie." *Sitzungsber. d. Würzb. Phys. Med. Gesellsch*, 1892 (*Neurol. Centr.*, p. 50, 1892). Two cases.

(29) Burgess. *Brit. Med. Jour.*, April 9, 1892. "Two cases of Friedreich's disease."

(30) Auscher. "Sur un cas de maladie de Friedreich avec autopsie." *Arch. de Phys. Norm. et Path.*, April, 1893, p. 340 (see also No. 4, same case).

(31) M'Caw. "Notes of a case of Friedreich's disease in a girl 8 years old." *Lancet*, vol. ii., p. 484, 1893. An isolated case.

(32) Krause. "Et tilfälde af Friedreich's sygdom." *Hosp. Tid.*, 1893, 4r., I., 30. Isolated case, classic sympt. (*Neurol. Centr.*)

(33) Chauffard. "Maladie de Friedreich avec attitude athetoïdes." *Sem. Med.*, vol. xiii., p. 409, 1893. Isolated case, marked jerky movements on exertion.

(34) Senator. "Über hered. ataxie." *Berl. Klin. Woch.*, vol. xxx., p. 489, 1893.

(35) Senator. Second article. *Berl. Klin. Woch.*, vol. xxxi., No. 28, 1894. Two cases in one family in those two papers.

(36) Anderson, Wallace. "Friedreich's ataxia." *Glas. Med. Jour.*, vol. xl., No. 3, 1893. Two cases, retained knee-jerks, mental impairment, slight athetosis.

(37) Mirto. "Atassia di Friedreich e atassia volgari." *Giorn Del. Ass. Dei Medici e Naturalisti*, Anno iv., puntati 3 e 4, 1893.

(38) Tresidder. "Three cases of hereditary ataxia." *Lancet*, vol. ii., p. 304, 1893.

(39) Wilson. "Notes on Friedreich's disease." *Birm. Med. Rev.*, vol. xxxi., October, 1893. Four cases, two isolated, all somewhat atypical.

(40) James. "Lecture on a case of Friedreich's ataxia." *Edin. Med. Jour.*, vol. xxxiv., p. 521, 1893. An isolated case; spinal curvature first symptom.

(41) Brock. "Three cases of Friedreich's ataxia." *Lancet*, vol. i., p. 139, 1893.

(42) Fry. "Two cases of Friedreich's ataxia." *Med. Fortnightly*, St. Louis, December 15, 1893. Typical; sisters.

(43) Benthall. "A case of Friedreich's ataxia." *Brit. Med. Jour.*, vol. i., p. 1,163, 1893.

(44) Lunz. "Ein Fall von Friedreich'scher Krankheit." *Deut. Med. Woch.*, August 17, 1893. A typical case with no nystagmus.

(45) Rauzier. "De la maladie de Friedreich." *Nouv. Montpelier Méd.*, vol. ii., pp. 725-748, 1893.

(46) Marie. "Sur l'hérédo-ataxie cérébelleuse." *Sem. Med.*, No. 56, 1893.

(47) Brissaud et Londe. *Rev. Neurol.*, 1894. One case of hereditary cerebellar ataxia.

(48) Mackenzie, H. W. G. "A case of non-hereditary Friedreich's disease." *Amer. Jour. of Med. Sci.*, vol. cvii., p. 371, 1894.

(49) Guizzeti. "Contributo illa anatomia patologica della malattia di Friedreich." *Il policlinico*, p. 438, 1894.

(50) Dreschfeld. *Lancet*, vol. i., p. 1,014, 1894. A short account of two isolated cases.

(51) Nammack. *Archiv. of Pediatr.*, New York, vol. xi., p. 386, 1894. An isolated case.

(52) Mackay. "The isolated type of Friedreich's disease." *Amer. Jour. of Med. Sci.*, vol. viii., p. 151, 1894.

(53) Schultze, Fr. "Uber die Fried. Krank. und ähnliche Krankheitsformen." *Deut. Zeitschr. fur Nervenh.*, vol. v., 1894. Heft 1-3, pp. 27 and 194. A criticism on Senator's case (No. 31 above), a re-examination of the cord of one of Friedreich's cases, with criticism of Déjerine and Letulle's observations, and a description of three new cases, which he calls a variety of Friedreich's disease (*ähnlich*).

(54) Besold. "Klinische Beiträge zur Kenntniss der Friedreich'schen Krankheit." *Deut. Zeit. für Nerv.*, vol. v., p. 157, 1894. Four isolated cases.

(55) Taylor, James. *Pract.*, November, 1894. Two cases, one with marked family tendency; the other isolated, with slight mental degeneration, and retained, though weak, knee-jerks.

(56) Krafft-Ebing. "Uber Fried. Krankheit." *Allg. Wiener Med. Z.*, No. 14, 1894. One case.

(57) Michell Clarke. "Case of Friedreich's disease, with necropsy." *Brit. Med. Jour.*, December 8, 1894.

(58) Schultze. "Erwiderung auf den zweiten Artikel von Senator." *Berl. Klin. Woch.*, No. 33, 1894.

(59) Tirasek. "Česopis lekařů ćeských." Praze. *Sajous' Annual*, 1894. One case of Friedreich's disease in a family with frequent intermarriages.

(60) Bouchaud. *Rev. Neurologique*, No. 1, 1894. (Ref. in Higier's article). Two cases allied to diplegia and Friedreich's disease.

(61) Londe. "De l'hérédo-ataxie cérébelleuse." *Thèse de Paris*, 1895.

(62) Guthrie. "Case of early Friedreich's ataxia." *Brit. Med. Jour.*, vol. i., p. 813, 1895. (Also in *Trans. Med. Soc., Lond.*, xviii., p. 382, 1894-5.) A boy of 9; no family predisposition, no nystagmus or speech defect.

(63) Nolan. "Three cases of Friedreich's ataxia, associated with genetous idiocy." *Dublin Jour. of Med. Sci.*, xcix., p. 369, 1895.

(64) Small, S. J. "Friedreich's ataxia, with cases." *Med. Rec.*, New York, xlviii., p. 85, 1895. Four advanced typical cases in a family, with strong neuropathic tendency. Onset at 13 or 14.

(65) König. "Cerebrale Diplegie der Kinder, Friedreich'sche Krankheit und multiple Sklerose." *Berl. Klin. Woch.*, xxxii., p. 716, 1895. A case like Friedreich's ataxia and disseminated sclerosis in some respects; also another case of hypoplasia of cerebellum and cord without inco-ordination.

(66) Bouchaud. "Maladie de Friedreich. Amblyopie, persistance du reflexe rotulien." *Jour. d. Sci. Med. de Lille*, No. 14, p. 1,313, 1895. The parents were first cousins. An isolated case.

(67) Stawell, R. K. "Two cases of Friedreich's disease." *Austral. Med. Jour.*, N. S., xvii., p. 45, 1895. Brothers, typical cases.

(68) Halbert. "Two cases of Friedreich's ataxia." *Clinique, Chicago.* No. 16, p. 636, 1895. Congenital syphilis, imbecility, spastic symptoms. Not examples of Friedreich's disease.

(69) Cardosi, S. "Contributo alla forme rudimentale del morbo di Friedreich." *Gazz. d. Osp. Milano*, No. 16, p. 158, 1895.

(70) Verhoogen. *Jour. de Med. de Chir. et de Pharm.*, Bruxelles, January 19, and March 9, 1895. Two typical cases of Friedreich's disease.

(71) Hoffmann. *Münch. Med. Woch.*, February 19, 1895. Two cases, one typically Friedreich's disease, the other doubtful. Epilepsy and mental weakness in this. Both isolated.

(72) Londe et Lagrange. *Annales de Med.*, Paris, March 7, 1895. "Two cases of Friedreich's ataxia." Sisters; athetoid attitude in walking, mental defect.

(73) Collins. *New York Med. Rec.*, December 21, 1895. "A contribution to the study of hereditary cerebellar and hereditary spinal ataxia, with the history of an atypical case of the first-named disease."

(74). Burr. "A contribution to the pathology of Friedreich's ataxia." *Univ. Med. Mag.*, Philadelphia (*Neurol. Centr.*, p. 376, 1895).

(75) Mackenzie, H. W. G. "Friedreich's disease." Article in *Med. Annual*, 1895. A short but valuable descriptive account, with a list of references.

(76) Baskett. "A case of Friedreich's disease." *Brit. Med. Jour.*, vol. ii., p. 749, 1896. No family affection.

(77) Dana, C. L. "A case of Friedreich's ataxia, with autopsy." *Postgraduate*, New York, vol. xi., p. 328, 1896.

(78) Higier. "Uber die seltene Formen d. hered. u. fam. Hirn und Rückenmarkskrankheiten." *Zeitschr. f. Nervenheilkunde*, vol. ix., pp. 1-61, 1896.

(79) Berdez. "Un cas isolé de mal. de Friedreich." *Rev. Med. de la Suisse rom.* (Genève), vol. xvi., p. 304, 1896.

(80) Fry, F. R. "Additional cases of Friedreich's ataxia." *Med. Rev.*, St. Louis, vol. xxxiv., p. 19, 1869. One an isolated case, the others sisters; of these one is very doubtful, the main feature being post-diphtheritic paralysis.

(81) Ormerod. "On Friedreich's ataxia." *Clin. Journ.*, London, vol. viii., p. 118, 1896-7.

(82) Riggs, C. E. "Two cases of Friedreich's ataxia." *North Western Lancet*, St. Paul, vol. xvi., p. 264, 1896. Cases not in same family, description very brief; one probably not correctly diagnosed (spastic gait, ankle and rectus clonus, but no knee-jerk).

(83) Rosenbaum. "Friedreich'sche Krankheit." *Deut. Med. Woch.*, vol. xxii., p. 483, 1896. Two cases of typical form; sisters.

(84) ZABLUDOWSKI. "Ein Fall von Friedreich'scher Krankheit. Behandlung durch Massage." *Berl. Klin. Woch.*, p. 757, 1896.

(85) TEDESCHI. "Die Friedreich'sche Krankeit, kritische und pathanat. Untersuchungen." *Beiträge z. Path. Anat., etc.*, vol. xx., pp. 51-84, Jena, 1896. A useful summary of autopsies. A careful pathological account of the case criticised above, and a bibliography.

(86) MAGNUS-LEVY. *Deut. Med. Woch.*, No. 28, 1896. *Ver. Beil.*, No. 19. Isolated case, doubtful. Details scanty.

(87) LEYDEN UND GOLDSCHEIDER. "Erkrankungen des Rückenmarkes." Wien, 1895-6.

(88) GOWERS. "Diseases of the nerves and spinal cord." London, 1892.

(89) VINAJ. "Malattia di Friedreich." *Osservazione Clinica. Estratto dalla Gazzetta Medica di Torino*, No. 51, 1894.

(90) EDINGER. "Eine neue Theorie uber die Ursachen einiger Nervenkrankheiten, insbesonders der Neuritis, und der Tabes." *Samml. Klin. Vortr.*, No. 106.

(91) RAYMOND. "Tabes juvenile et tabes héréditaire." *Le Progrès méd.*, 1897. No. 32 and 33.

A Case of Friedreich's Ataxia, with Autopsy and Histological Examination. By JULES SIMON. (*Le Progrès Méd.*, 1897, September 4, No. 36.)

JULES SIMON relates the following case :—Boy, aged 10, only child, admitted to Enfants-Malades in 1889 for pains in legs. Nothing hereditary of importance. Had pains in legs at 2½. dysentery at 5, after which he walked badly. In 1891, following was observed : strumous aspect, chronic blepharo-conjunctivitis, old keratitis, not intelligent-looking, speech slow, jerky. No nystagmus, club-foot, scoliosis, sensory troubles or sphincter affection. Knee-jerks absent. Standing difficult, especially if eyes shut. "Drunken" gait, often falls. Diagnosis of Friedreich's ataxia, confirmed by Joffroy. In 1896, slight tremor in head, jerky and fibrillary movements in hands ; motion and sensation normal in upper extremities. Lower limbs cannot support him. Atrophy of calves and muscles of soles, club-foot, no knee-jerks, normal sensibility, cold feet. Pupils normal, transverse nystagmus on lateral fixation. Tends to slaver, especially on laughing, slight deviation of mouth to right. Cannot whistle now but could formerly. No scoliosis. The pains he has from time to time consist in intense heat and pricking in calf regions. Intelligence mediocre. Often has headaches followed by vomiting preceded by giddiness. Incontinence of urine frequently.

Death sudden and unexpected.

Post-mortem.—Normal naked eye appearance of cerebrum and cerebellum; weight of latter normal. No histological examination (unfortunately *Rep.*).

Examination of cord by Philippe (three months in Müller's fluid, Weigert-Pal stain).—I. Posterior columns show loss of myeline sheaths. (1) Sacral region, level of third sacral root: rootzones more affected than the zones of endogenous fibres, the short commissural fibres; myelinic reticulum of posterior horns very poor. (2) Lumbar region: rootzones specially affected, also the centrum ovale of Flechsig; cornu-commissural zone nearly intact; myelinic reticulum of posterior horns affected as in sacral region. (3) Dorsal region: decoloration of posterior columns at maximum in upper dorsal region; spec. the lumbo-sacral fibres, near the posterior fissure; the myelinic reticulum in Clarke's column scanty. (4) Cervical region (sixth root): Goll's column nearly completely decolorized; the postero-external column also well decolorized but less so than in dorsal region. (5) Bulbar region: fasciculi gracilis and cuneatus much decolorized, the former more, representing as it does Goll's column.

II. Posterior roots, loss of myeline everywhere, more so where the adjacent posterior columns are most affected.

III. Lateral columns, affected all along cord, but less so than posterior column; pyramidal tract and cerebellar tract affected; direct pyramidal tract in upper dorsal and cervical regions.

Where the nerve tubes have gone, their place is taken by a dense sclerosed neuroglia, arranged in places in whorls (both in posterior and lateral columns); connective tissue thickening of pia mater over posterior aspect; considerable diminution in number and volume of cells in posterior horn and at base of anterior horn; also of column of Clarke in lumbar and dorsal regions.

Conclusion.—The cord is the seat of a combined sclerosis affecting the posterior columns, the pyramidal bundle and the direct cerebellar tract. This corresponds fairly well with the lesions hitherto found in cases of Friedreich's disease. In addition to the combined sclerosis of the white matter, there is a lesion of the gray substance.

LESION OF THE SUPERIOR PARIETAL LOBULE.

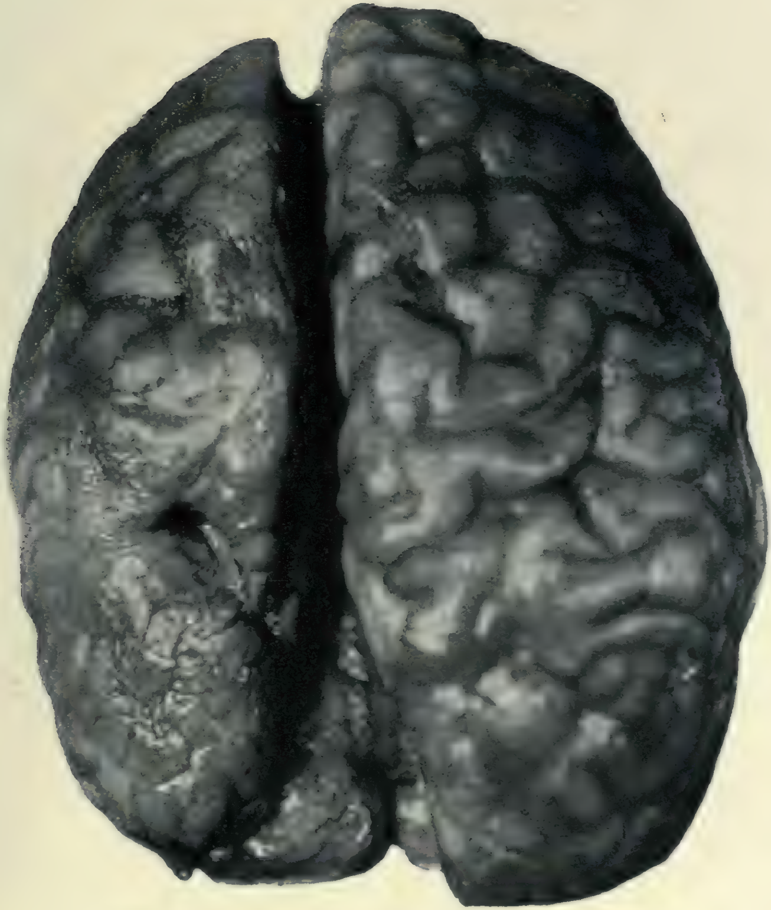
BY CECIL F. BEADLES.

ANY clinical evidence that may throw light on the areas of the cortical centres cannot but be of interest and importance. The following case has reference to the superior parietal lobule. A further point that makes this of some interest is the fact that definite lesions (except for congestion) of the brain of epileptics dying insane are but seldom seen.

The patient, 11445, was a youth who was admitted into Colney Hatch Asylum on November 25, 1891. He was then aged 18, and was the subject of epilepsy. His mother had died insane. The certificate stated that he was dull and of heavy expression, that he used bad and threatening language and had shown violence, and that he had been subject to fits since childhood. During the year 1892 he had frequent and severe fits, which occurred almost daily; he was weak-minded with impaired intelligence and almost incoherent, and was frequently violent in his behaviour. His health was fair and he was well nourished; was of clean habits but never occupied. In 1893 he continued much the same, although on the whole of quieter behaviour. Fits varied from several a week to three or four daily. They occurred both day and night. Some weakness of the right arm was noted.

In 1894 fits occurred daily. He was an impulsive and violent epileptic, continually quarrelling and fighting; his mental state was that of an imbecile. No change occurred the following year. Bromide of potassium was tried from time to time, but appeared to have but little effect on the epileptic paroxysms.

Up to January 6, 1896, the patient had two or three



Lesion of left superior parietal lobule. (*From a photograph.*)

fits almost daily. Then for twenty-four hours he had a large series of severe fits, more than thirty in number. The following day he had four more fits, and he died on January 8 after another succession of fits without regaining consciousness. Between the date of admission and death the patient had 3,360 fits (a rapid succession being counted as one); of this number 1,282 occurred during the last year of his life.

The special point of interest in the case is in reference to his right leg. The right ankle was drawn up and the toes were contracted. When he walked he dragged this foot and bore his weight on the left leg. No reference is made to this condition on the patient's admission, but it is probable that it then existed.

When he attempted to run, which almost invariably preceded a fit, his right foot seemed to trip him up. As he went off into the fit he would scream and roll about on the ground, crying out several times "Oh my leg, oh my leg." As he recovered from it the toes of the right foot were more contracted than usual.

At the autopsy the abdominal and thoracic viscera were normal with the exception of the heart, which was slightly hypertrophied with some excess of fat externally, and a small amount of atheroma above the aortic valves. It was found that the skull was greatly thickened throughout. The external conformation and measurements of the cranium were of fair size, but the internal capacity was distinctly small: all bony prominences within the fossæ were very marked. The dura mater was inherent to the calvaria and a considerable excess of fluid existed.

The whole brain was reduced in size, but the left cerebral hemisphere was much more contracted than the right. The left side was covered by a thickened milky pia arachnoid membrane, while the right was not thickened but was congested. The pons and medulla appeared smaller on the right side, and the right cerebellar lobe also seemed slightly reduced in size; the membrane over both these lobes was congested but not noticeably thickened. The weight of the whole brain before examination was 36½ ounces.

A localised lesion existed on the upper and hinder part of the parietal lobe of the left cerebral hemisphere. Situated in the hinder part of the superior parietal lobule, immediately in front of the parieto-occipital fissure, was a depression on the surface of the brain of the size of a fair sized marble. This was filled in the recent state by fluid in the meshes of the thickened pia mater. We have no evidence as to the cause of the lesion; no injury is known to have occurred. This lesion evidently involved the grey cortex. It is the site representing the area attributed to the centre for movement of the small toes, and this is fully borne out by the clinical evidence given above.

Gowers ("Diseases of the Nervous System," 1893, vol. ii., p. 17), in his explanation to a diagram illustrating the cortical centre for the leg, suggests that the centre does not extend as far back as the parieto-occipital fissure on the outer aspect of the hemisphere, but it will be seen in this case that the atrophy of the convolution reaches that point, and also extends down in front of that fissure on the inner surface of the hemisphere.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At the Clinical Meeting of the Society, held at 11, Chandos Street, W., on Thursday, February 24th, the following cases were shown :—

Dr. Donkin.—(1) A child, about 4 years old, with spasm of the extremities, more marked in the legs than in the arms, especially, though not entirely, occasioned by attempts at using them; associated with great coldness and blueness of feet. In some respects resembles a case of Thomsen's disease. The child was quite well and walked without difficulty until aged 2½ years. No history of any morbid heredity. There is no wasting or other abnormality discoverable on examination of the nervous, muscular, and other systems. (2) A man, aged about 50, with hemianæsthesia (including special senses) alternately appearing and disappearing on the same side, associated with weakness of the affected limbs. (The man is readily thrown into the hypnotic sleep.) The right field of vision is much contracted and the left almost *nil*. He protrudes his tongue towards the right, the left side of the tongue being markedly humped and the right angle of the mouth drawn downwards and to the right. When taste is being tested the tongue is protruded normally, and straight. There is probably a certain element of malingering in this case, but at first the symptoms were clearly genuine. (3) A case of paralysis agitans in a man aged 43. The tremors are only noticeable on use of the limbs. The motor symptoms and difficulty in walking were preceded by a long period of much aching pain in back and legs. The symptoms of propulsion and retropulsion are strongly marked, and the facial expression and bodily attitude are characteristic.

Dr. Mott.—A woman, aged 50, married, seven children, two born dead at full term. Present illness commenced four years ago, immediately after the menopause, with numbness in the hands. The hands became swollen, and she had frequently pins and needles in them. March, 1897, numbness occurred in the chin, followed by pins and needles, and the pain and numbness gradually spread to the lips and cheeks; the tongue also felt large, and the skin of the lower part of the face tense and swollen, with diminished sensation. Her hands and feet are swollen and somewhat cyanosed; they feel numb, and she does not feel well in the tips of the fingers. There is *diminished sensibility* to pain, pressure and thermal stimuli over the structures supplied by the two inferior divisions of the fifth nerves; the ophthalmic branch of the fifth is not affected. There is loss of taste over the anterior two-thirds of the tongue to salt, acid or sweet fluids; at the back of the tongue she tastes quite well. She did not smell assafœtida or ammonia. There was an erythematous rash over the upper part of the chest and neck.

Dr. HERBERT HAWKINS.—A lad, aged 18. About four years ago noticed that he had to lift his feet high in order to clear the ground in walking, and two years later noticed wasting of muscles of both thumbs. Nothing in family or personal history to throw light on this. Now, has great wasting and complete paralysis of muscles supplied by anterior tibial nerves on both sides, with slighter affection of peronei: great wasting of thenar and hypothenar muscles, with slight wasting and weakness of extensor muscles in forearms. No affection of any form of sensation. Fibrillar contractions in quadriceps extensor on both sides. Knee-jerks, present last November, disappeared in December. Well-marked nystagmus. Electrical reactions; true R.D. in left posterior inter-osseous group of muscles, and in anterior tibial groups on both sides; reduction of irritability to both currents in right posterior inter-osseous group, in intrinsic muscles of hands and in peronei.

Dr. BEEVOR.—Case of syphilitic meningitis. A man, aged 25. Syphilis and gonorrhœa twelve months previously. Onset in August, 1897, with malaise, severe headache, vomiting, followed in five days with severe pains in both arms and difficulty in moving them. No anæsthesia, no tingling, Glycosuria for a week. No pyrexia. At present the following muscles are paralysed on both sides:—Trapezius below level of second dorsal spine, exposing rhomboids which can be seen; serratus magnus; deltoids, except anterior and posterior parts, on the right. The following are weak:—Latissimus dorsi, pectoralis major, triceps. At rest the scapula was tilted, bringing the lower end nearer to the spinal column; no power to advance or abduct humerus beyond horizontal line, the latter being performed by supraspinatus acting without the deltoid.

Dr. **Leonard Guthrie**.—Unilateral syringo-myelia. Female, aged 29 years. Thermo-anæsthesia, analgesia of left side of head, neck and face, with unilateral seborrhœa capitis; nystagmus, paralysis of left side of palate and of left vocal cord. The bulbar symptoms are of two years' duration. They were formerly associated with analgesia and thermo-anæsthesia of the left upper extremity.

Dr. **Stephen Mackenzie**.—"A Case of Nodding Spasms (*Spasmus nutans*)." The patient is an infant, one year of age, who has not yet erupted any tooth. The spasms consist of combined nodding and rotatory movements of the head. As usual in such cases, these movements are combined with nystagmus, in this case in a lateral direction. The child is well nourished, and in other respects appears well.

Dr. **Ormerod**.—General athetosis, with double equino-varus, of uncertain origin, in a lad aged 19. Duration of disease five years.

Movements.—Slow, irregular wriggling movements of head, trunk and limbs, producing varied positions of the body, and occasionally throwing patient out of bed or chair. Larger joints more affected than small. Movements constant, except that they intermit temporarily when he uses limbs voluntarily, and that they cease during sleep; physiognomy peculiar; occasional slow grimacing; speech slovenly, nasal, sometimes almost unintelligible; breathing irregular and grunting.

Gait and condition of feet.—Double talipes equinus and pes cavus; dorsiflexion of metatarso-phalangeal joints of toes; contraction of calf-muscles; walks entirely upon his toes. Apparently intelligent, and has always been so; passed fifth standard at school.

History.—Present condition developed without assignable cause and gradually, at age of 14, after leaving school; movements said at first to have occurred only during voluntary actions. Father said to be nervous and alcoholic; family history otherwise negative.

Dr. F. E. **Batten**.—"Friedreich's Disease and Mental Defect." Boy, 11½ years old; eldest of family of seven; natural labour; late in learning to walk; late in learning to talk; always backward; unsteadiness in walking (increased during the last two years); dull boy; ataxia; speech defect (high palate); nystagmus (not constant); inco-ordination of arms; knee-jerks absent, pes cavus. Mother had epileptic fits before marriage. No hereditary history.

Dr. Aldren Turner.—A youth, aged 22, who had convulsions in infancy, but no subsequent fits. The present condition is as follows: — Complete diplopia facialis; convergent strabismus, owing to paralysis of both external recti muscles; double club-foot; a spastic gait and exaggeration of the knee-jerks; defect in the finer movements of the hands (one year ago there were irregular athetoid movements of the fingers); myopic astigmatism; no affection of sensation.

The Society held its first meeting out of London on Saturday, April 23rd, when, in response to an invitation from Dr. Alex. Hill, Vice-Chancellor of the University of Cambridge, a meeting was held in the Physiological Laboratory of that University, at 4.30 p.m., when the following communications were made:—

Dr. Alex. Hill.—"Some Specimens of Granules."

Professor Macalister.—"The Race-Characters of Australian Brains."

Dr. Langley.—"Some points relating to the Sympathetic System."

Dr. Gaskell (for Miss Alcock).—"The Distribution of the Facial Nerve in Ammocœtes."

Dr. F. E. Batten.—(1) "Nerve Terminations in Muscle Spindles." (2) (for Dr. Sihler, of Ohio).—"Motor Nerve Termination *within* the Muscle Spindle."

Dr. Mott.—"Degeneration of the Neuron."

Dr. Langley's communication was an experimental demonstration, and the other papers were illustrated by lantern slides and by macroscopic and microscopic specimens.

At the conclusion of the meeting, members and visitors were entertained at dinner by the Vice-Chancellor, in Downing Hall.

BRAIN.

PART II., 1898.

Original Articles and Clinical Cases.

CONTRIBUTIONS TO THE STUDY OF SOME OF THE AFFERENT AND EFFERENT TRACTS IN THE SPINAL CORD.

BY J. S. RISIEN RUSSELL, M.D., F.R.C.P.

Assistant Physician to University College Hospital, and Pathologist to the National Hospital for the Paralysed and Epileptic, Queen Square.

INTRODUCTION.

These studies deal more especially with :—

(1.) The posterior columns.

(2.) The direct cerebellar tract.

(3.) The afferent antero-lateral tract.

(4.) The pyramidal system, including the crossed, the direct anterior, and direct lateral (homolateral) pyramidal tracts.

(5.) The efferent antero-lateral tract.

(6.) Certain other efferent internuncial tracts.

They are based on investigations of the spinal cords of the following cases in man :—

(1.) Degeneration of the first sacral roots in a case of alcoholic neuritis.

(2.) A tumour involving chiefly the seventh cervical root on one side, and a limited portion of the lateral region of the cord on the same side.

(3.) An intra-medullary tumour of the lower part of the lumbo-sacral cord.

(4.) A complete transverse lesion of the spinal cord at the level of the sixth and seventh cervical segments, the result of a fracture dislocation.

(5.) A case in which a large tumour was removed from one cerebral hemisphere.

(6.) A large cyst of the centrum ovale of one cerebral hemisphere, the result of the degeneration of a malignant tumour.

My sincere thanks are due to Sir William Gowers for allowing me to make use of Case 1, to Dr. Beevor for Case 2, to Mr. Victor Horsley for Cases 3, 4, and 5, and Dr. Charlton Bastian for Case 6.

I propose to first give a brief account of the chief points in each case, as they concern our present study, and then to discuss the facts which appear to be the outcome of the investigations collectively, and in conjunction with previously ascertained facts of a like character.

Degeneration of the First Sacral Roots in a Case of Alcoholic Neuritis.

In this case sections prepared by Marchi's method show degeneration of both first sacral roots, but that on the one side is only slight, whereas the other root is considerably, though not completely degenerated. On entering the cord the fibres at first hug the grey matter of the posterior horn, being limited to the dorsal half of that part of the posterior columns which borders on the grey matter (see fig. 1). They, however, quickly become scattered through the posterior columns, so that sections from the 5th lumbar segment show that no part of these tracts on the side of the more degenerated root is entirely free from degenerate fibres, excepting the cornu-commissural zone and septo-marginal tracts, though these are still very much more numerous in the dorsal half of the columns, and especially in the outer part next to the grey matter. No degenerate fibres are seen either in the cornu-commissural zone, that is, the ventral part of the posterior columns bordering on the posterior commissure, septum and cornu, and to which the name cornu-commissural tract has been given by Marie, or in the septo-marginal tract, so named by Bruce and Muir in consequence of its immediate relationship to the posterior median septum.

There is distinct degeneration of many of the reflex collaterals, numerous degenerated fibres of this kind being seen coursing through the grey matter to the ventral horn of the same side, both in the 5th lumbar and first sacral segments of the cord; but none pass to the ventral horn of the opposite side, nor do any of the fibres pass beyond the grey matter of the same side as the affected posterior columns, to reach the white matter of the ventro-lateral region of the cord. A few collaterals are, however, seen passing across the base of the posterior horn towards the lateral column of the same side, without any degenerated fibres being actually seen in this column. There is also degeneration of some of the fine collaterals in the external part of the gelatinous substance of Rolando. No degenerated fibres can be seen passing by any route to the opposite posterior columns, and those seen in them clearly belong to the opposite posterior root, which is slightly degenerated.

In the 3rd lumbar segment of the cord the distribution of the degeneration differs from that above described; there is degeneration of all parts of the postero-internal column, including the cornu-commissural zone, and there is also degeneration of the ventral third of the postero-external column, and of a dorsal zone of it at the periphery of the cord, not quite amounting to a third of the dorso-ventral area of the tract. The middle third of the postero-external column is free from degeneration, especially that part of it which borders on the substantia gelatinosi Rolandi. No degenerate fibres are seen passing to the ventral horn of the same side, or to any other part of the grey, or white matter of the cord, other than the posterior columns as already described.

In the thoracic region the precise distribution of the degenerated fibres naturally varies with the level at which they are seen; but the degeneration tends more and more to limit itself to the postero-internal column, and more and more to the dorsal part of this, according to the well established law with regard to fibres derived from the caudal segments of the cord. Thus it comes about that less and less degeneration is seen in the postero-external column, and in the cornu-commissural zone, as we pass towards the cephalic end of the cord. No degenerate fibres are seen passing to the grey matter of the ventral horn, or any other part of the cord.

The degeneration as seen in the cervical cord is limited to the dorsal two-thirds of the postero-internal column, the area thus occupied being triangular in area with its apex abutting on the posterior median septum ventrally, and the base being situated dorsally at the periphery of the cord, at which part the degeneration occupies the whole transverse area of the postero-internal column. All other parts of this column are free from degeneration, as is the postero-external column. No degenerated fibres are seen passing to any of the other tracts of the cord, nor can any be traced to the grey matter of either ventral horn or elsewhere.

Tumour Involving Chiefly the Seventh Cervical Root on one Side, and a Limited Portion of the Lateral Region of the Cord of the Same Side.

This is a case in which a large sarcoma in the neck extended into the neural canal along the 7th cervical root, which it destroyed, and to some extent pressed on the 6th and 8th cervical roots, without, however, either involving or destroying them. The growth also pressed upon the right half of the cord, so as to push it over to the left side of the neural canal, and at the same time destroyed a limited area of the lateral region of it, corresponding to the areas occupied by the direct cerebellar and crossed pyramidal tracts on the right side. Otherwise the cord, as seen on transverse section, was intact.

The degenerations seen in segments of the cord above the lesion consist in a well-marked band of degeneration

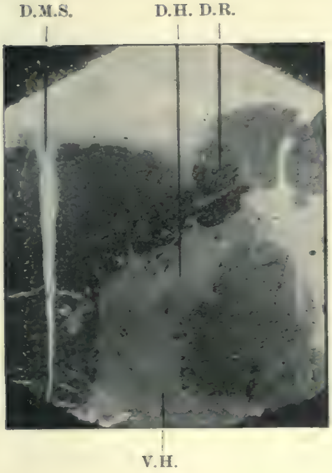


FIG. 1.

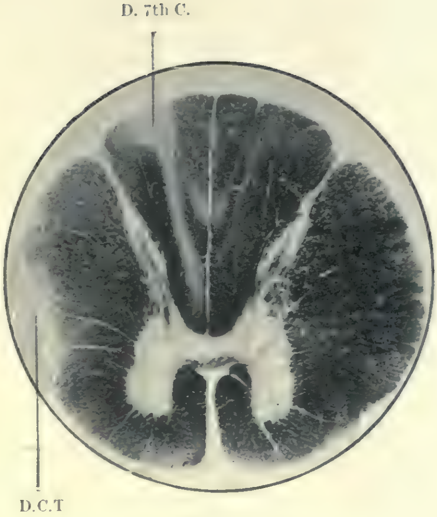


FIG. 2.

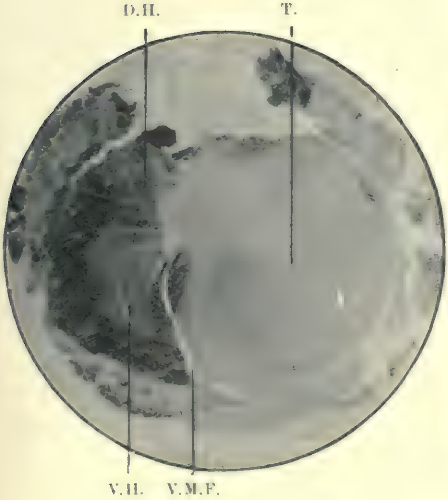


FIG. 3.

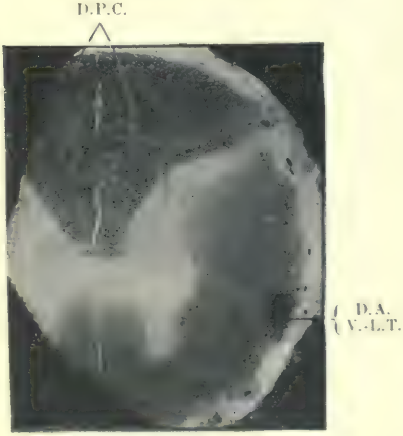


FIG. 4.

which at the level of the 3rd cervical segment extends almost from the posterior commissure to the dorsal periphery of the cord. The ventral end of this degenerated area is separated from the posterior commissure by a narrow band of healthy fibres of the cornu-commissural zone. The dorsal end of the degenerated area at the periphery of the cord is somewhat abruptly widened out, for about a fifth of its total length, to about four or five times the breadth of its ventral four-fifths, the ventral third being in its turn slightly wider than the portion separating it from the wide peripheral dorsal area (see fig. 2). This degenerated band is accurately limited to the inner border of the postero-external column throughout the greater part of its length, coming to within a short distance of the anterior fourth of the posterior median septum from which it is only separated by a very narrow band of healthy fibres in this part. The band of degeneration maintains this position in its further course to the medulla without encroaching on the postero-internal column.

In addition to this well-marked band of degeneration extending along the inner border of the postero-external column on the right side, and separated from the postero-internal tract by the septum intermedium, there exists, on each side of the posterior median fissure, a small area of degeneration, absolutely symmetrical, and limited to the postero-internal columns. These degenerations are situated at the ventral end of the dorsal half of the postero-internal columns, and are so placed that their ventral extremities, which are broader than their dorsal, abut on the posterior median fissure. Their dorsal, narrow, or tailed ends, on the other hand, are situated at some distance from the posterior median septum, so that these degenerated areas lie somewhat obliquely across the postero-internal columns.

The only other tract degenerated above the level of the lesion is the direct cerebellar tract on the side of the damage to the spinal cord (see fig. 2).

Below the lesion, on the other hand, as seen in sections caudal to the 3rd thoracic segment, the only tract found degenerated is the crossed pyramidal tract on the side of the tumour, and this could be traced to the most caudal end of the lumbo-sacral cord.

INTRA-MEDULLARY TUMOUR OF THE LOWER PART OF THE LUMBO-SACRAL CORD.

In this case the tumour involved the whole of the conus terminalis and extended up into the lumbo-sacral cord to a greater height on one side than on the other. At the lower part of the sacral cord it was more or less centrally situated with a margin of spinal cord tissue all round its periphery, but even here there was a distinct tendency for the one lateral half of the cord to escape to a greater extent than the other. By the time the level of the 4th lumbar segment was reached the evidence of the greater involvement of one half of the cord was most pronounced, nearly all of the grey matter on one side having escaped together with all the white matter, except both posterior columns, the direct pyramidal tract and part of the adjacent ground bundles (see fig. 3). The destruction of the other half of the cord was even more complete than at more caudal levels, in that the growth reached to the periphery of the cord, except at one or two small points.

The 3rd lumbar segment, though not itself actually involved by new growth on either side, was nevertheless swollen and softened on one side, probably in consequence of interference with its vascular supply by the growth in the adjacent segment. The swollen, softened condition corresponded to the side on which the tumour reached its highest level.

As far as can be determined the 2nd lumbar segment is free from any morbid change other than the ascending degeneration of afferent tracts, and in this lies the chief interest of the case from our point of view, in that both the postero-external and postero-internal columns are degenerated on both sides of the cord, while with the exception of a few scattered degenerated fibres the ventro-lateral ascending tract is intact on one side, but is distinctly degenerated on the opposite side, as shown in fig. 4, which is from a photomicrograph of a section from the 11th thoracic segment of the cord. The side on which this band of degeneration exists in the region of the ventro-lateral ascending tract corresponds to that on which the tumour reaches its highest level.

The degenerations though obvious in sections stained by Pal's method, being recent in character were very much more pronounced as seen in sections prepared by Marchi's method, which revealed nerve fibres in a recent state of degeneration in all parts of the posterior columns. The latter method also allowed of degenerated fibres being traced from the posterior columns to other parts of the cord, but we do not propose to enter on the question of their distribution on the present occasion. Apart from degenerated fibres of this kind, the degeneration of the posterior columns on both sides, and the ventro-lateral ascending tract on one side, the other tracts at this and higher segments of the cord are preserved.

Before leaving this part of our subject we may state that examination of the *cauda equina* showed that while many bundles of degenerated fibres existed in it, a considerably larger number of bundles were composed of normally medullated fibres. Indeed, the degenerated bundles were not more than could be accounted for by those proceeding from the destroyed segments of the cord, without supposing that the growth in the cord had caused any direct damage by pressure of the fibres of the *cauda equina*, of which there was also no other evidence.

Complete Transverse Lesion of the Spinal Cord, at the Level of the Sixth and Seventh Cervical Segments, the Result of a Fracture Dislocation.

In this case the spinal cord was completely destroyed at the level of the 6th and 7th cervical segments, so that no trace of grey or white matter could be found at these levels two years after the accident, except that in the posterior part of the cicatrix some posterior root fibres (? regenerated) could be seen.

Immediately on the cephalic side of the lesion there is complete sclerosis of the posterior columns, Lissauer's tract, the direct cerebellar and afferent ventro-lateral tracts on both sides. As we pass further towards the cephalic extremity, in accordance with the well-established rule, the degeneration in the posterior columns diminishes in area in consequence of the incoming roots above the level of the lesion supplying normal fibres to the outer zone of the postero-external column which borders on the grey matter of the posterior horn as shown in fig. 5, taken from a section at the level of the 4th cervical segment. These normal fibres must of necessity be supplied by the 5th cervical and other roots nearer the brain in the series; and it is worthy of note that the incoming normal fibres are limited to the postero-external column, even at the first cervical segment of the cord.

The degeneration of the direct cerebellar tract calls for no comment, except that it is interesting to note how closely the degenerated area in this case corresponds to that in the case in which this tract was interrupted on one side by a tumour at the 7th cervical segment. I wish, however, to call special attention to the position occupied by the sclerosed ascending ventro-lateral tract of Gowers, which is situated about midway between the periphery of the cord and the ventral horn, and while it merges dorsally into the area occupied by the direct cerebellar tract, there is no evidence that it ever comes to the periphery of the cord (see fig. 5).

Immediately caudal to the lesion there is marked sclerosis of the crossed and direct pyramidal tracts, the comma tracts of Schultze in the postero-external columns, and the ventro-lateral efferent tract. The only point to which I wish to call special attention in this connection is that the ventro-lateral efferent tract occupies the extreme periphery of the cord (see fig. 6), and this contrasts markedly with the afferent ventro-lateral tract, which is separated from the periphery by a well-marked band of normal fibres; the fibres of the efferent tract in fact.

In that the case was one of such long standing the Marchi method was not available for tracing the degenera-

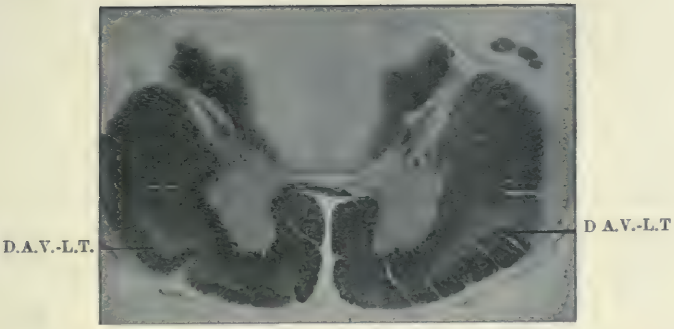


FIG. 5.

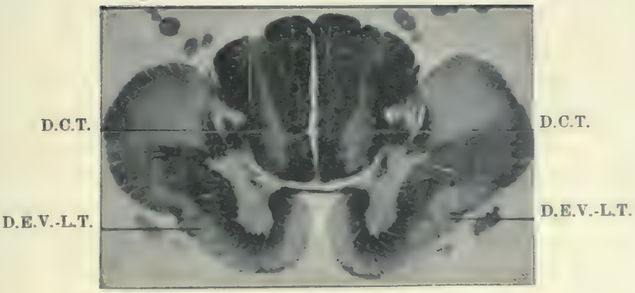


FIG. 6.

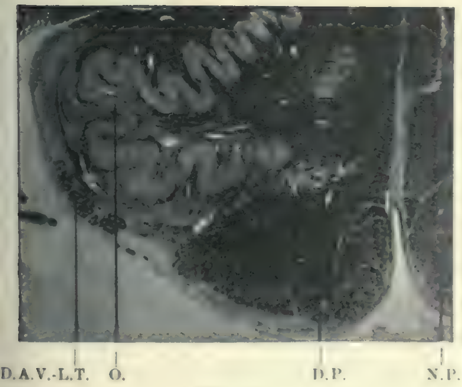


FIG. 7.

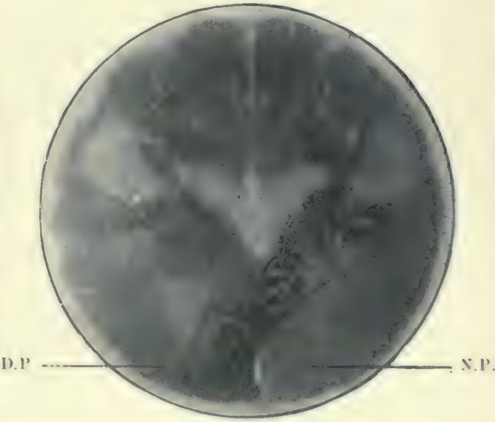


FIG. 8.

tions, which were accordingly traced by Pal's method. This allowed of the crossed pyramidal tracts being traced to the caudal extremity of the cord ; but the sclerosis of the direct pyramidal tract can not be made out with certainty farther than the fourth lumbar segment, where it is very slight, while the sclerosis of the ventro-lateral efferent tract is still distinct at this level, but occupies only a limited part of the periphery of the cord close to the anterior median fissure.

The comma tract in the postero-external column can be traced by this method with certainty to the 6th thoracic segment, so that it extends, as a well-defined tract, through seven segments of the cervico-thoracic cord. From its appearance at the 6th thoracic segment, I have little doubt that had the Marchi method been available it would have been possible to trace this tract much farther caudally.

Case in which a Large Tumour was Removed from one Cerebral Hemisphere.

A large sarcoma had been removed from the right cerebral hemisphere five weeks before the patient died. The area of cerebral cortex excised in its removal was such that the lower 5 cm. of the ascending frontal, and 2 cm. of the ascending parietal were left intact ; the hinder part of the superior frontal was slightly implicated, but both the other frontal convolutions escaped, the anterior boundary of the removed area being the precentral sulcus. The posterior boundary corresponded to about the posterior end of the Sylvian sulcus, on the external aspect of the hemisphere, while, on the mesial aspect, this limit was marked by the posterior end of the calloso-marginal sulcus, at which point the dura was adherent. Otherwise 1½ cm. of the upper end of the ascending frontal, and ascending parietal convolutions, together with the superior parietal, were intact. A vertical section into the hemisphere in the direction of its long axis, and about 2¼ cm. from the middle line, showed that the region from which the tumour had been removed embraced an area 7 cm. in the antero-posterior direction and 4 cm. in the vertical, limited to the white matter as far as the surface grey matter was concerned, but reaching to the junction of the white matter and the grey bounding the hinder end of the fissure of Sylvius. There was in addition some softening in the region of the optic thalamus.

As the patient died five weeks after the tumour was removed, the case was one in which the Marchi method proved of the greatest possible advantage in tracing the degenerations consequent on the cerebral lesion. All that we propose to do, on the present occasion, is to consider the mode of behaviour of the pyramidal system, and certain other efferent tracts which degenerated in the spinal cord.

Before the decussation of the pyramids at the caudal end of the medulla there is, of course, pronounced degeneration of the pyramid on the side of the cerebral lesion (see fig. 7), while the opposite pyramid is absolutely intact.

In addition to this degeneration of the pyramid, degenerate fibres are seen in the inter-olivary stratum, on the side of the lesion occupying a limited part of this region, which abuts on the dorsal aspect of the pyramid. Another and quite distinct tract of degeneration seen on the side of the lesion is one which occupies the ventral margin of the medulla, just outside, and opposite to the ventro-external angle of the inferior olive. This is a well-defined band of degeneration, and is quite distinct and separate from the degenerated pyramid, lying between the external arcuate fibres as they course round the medulla opposite the inferior olive (see fig. 7). It has not been found possible to establish with certainty a connection of this tract with any of the degenerated fibres met with in the pons; but on the other hand, no secondary lesion, meningitic or otherwise, has been found to account for the degeneration of these fibres.

Throughout the whole length of the decussation successive sections reveal a thick band of degenerated fibres, stained black by the Marchi method, streaming across the medulla oblongata to reach the opposite crossed pyramidal tract. So pronounced is this band that it appears as if the whole of the fibres of the degenerated pyramid at the particular level under observation were passing to the opposite crossed pyramidal tract (see fig. 8). A few scattered fibres are, however, seen passing from the degenerate pyramid to the lateral pyramidal tract of the same side, especially towards the cephalic end of the decussation, but the number of these is small compared to what one would have expected with so extensive a degeneration of the pyramid.

It is impossible to distinguish the degenerate fibres that were seen in the inter-olivary stratum from those of the pyramid, at this level; but throughout the caudal end of the medulla there is seen a distinct tract of degenerated fibres, separate from the pyramid, and occupying the lateral region of the medulla opposite the ventral tip of the anterior horn (see fig. 9). This is the same tract of fibres which was seen opposite the ventro-external angle of the inferior olive before the decussation; but whereas in that situation it was a narrow band of some length, here it occupies a more or less oval area.

A well-defined group of degenerated fibres occupies the dorsal part of the otherwise unaffected pyramid (see fig. 10), which structure, it will be remembered, was quite free from any degeneration above the decussation. This bundle of fibres lies between the root of the ventral horn and the decussating fibres derived from the degenerated pyramid.

In the cervical cord, as seen in sections at the 5th segment, the efferent tracts found degenerated are both lateral pyramidal tracts, the direct pyramidal tract on the side of the cerebral lesion, a tract at the periphery of the ventral part of the cord, opposite the ventral horn, and another collection of fibres on the inner side of the ventral horn of the side of the cord opposite to the cerebral lesion. Of these degenerations by far the most extensive is, of course, that of the crossed pyramidal tract on the side opposite to the lesion. Degenerated fibres occupy a far wider area than that usually assigned to the fibres of the crossed pyramidal tract which are derived from the cerebrum. The peripheral region of the cord in the positions of the direct cerebellar, and ventro-lateral afferent and efferent tracts is spared, but degenerated fibres otherwise occupy the whole of the lateral area of the cord up to the grey matter of the posterior and anterior horns, the lateral limiting layer being crowded with degenerated fibres. Moreover, the degenerate fibres are seen ventrally to the supposed limits of the pyramidal tract chiefly in that part of the white matter which abuts on the anterior horn, and they form a well-marked band which extends round in the region of the anterior ground bundles on the ventral side of the horn so as almost to meet the group of degenerated fibres situated on the inner side of the horn, though there is a distinct gap between these two areas of degeneration. A few scattered degenerated fibres are seen at the periphery of the cord in the dorsal part of the afferent and efferent ventro-lateral tracts, extending as far ventrally as to be on a level with the tip of the ventral horn.

The degeneration seen in the lateral pyramidal tract on the side of the lesion is scanty, scattered and much more limited as regards area than that of the opposite lateral tract. There is extensive degeneration of the direct pyramidal tract on the side of the lesion, limited chiefly to its dorsal two-thirds, while its ventral third is comparatively free from degenerated fibres as shown by the Marchi method.

The tract at the periphery of the cord, opposite the tip of the ventral horn on the side of the lesion, is composed of a limited number of degenerated fibres (see fig. 11), scattered and forming more of a band as they did on the cephalic side of the decussation of the pyramids, rather than a more or less oval column as was the case at the caudal end of the medulla oblongata.

The group of fibres on the inner side of the opposite anterior horn is scanty, and the fibres are scattered; they occupy the territory of the anterior ground bundles close to the grey matter, and of that part of it bordering on the ventro-internal tip of the ventral horn (see fig. 12). The portion of the ground bundles bordering on the root of the ventral horn and anterior commissure is free from degeneration, as is the direct pyramidal tract on this side.

The degenerate fibres in the crossed pyramidal tract of the side opposite to the cerebral lesion can be traced

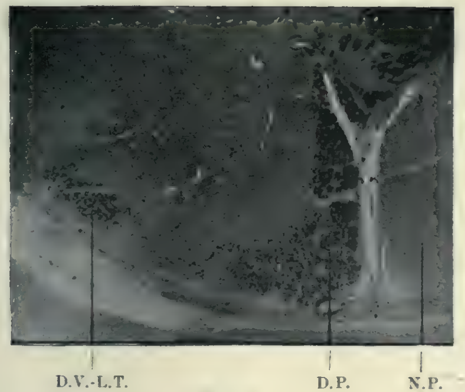


Fig. 9.

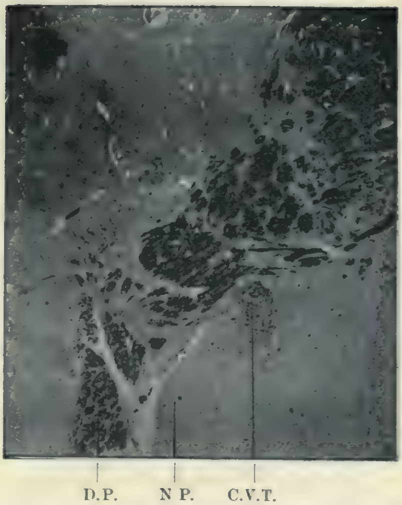


Fig. 10.

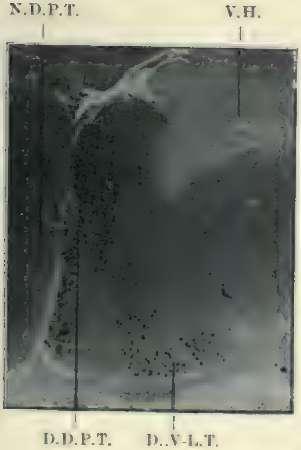


Fig. 11.

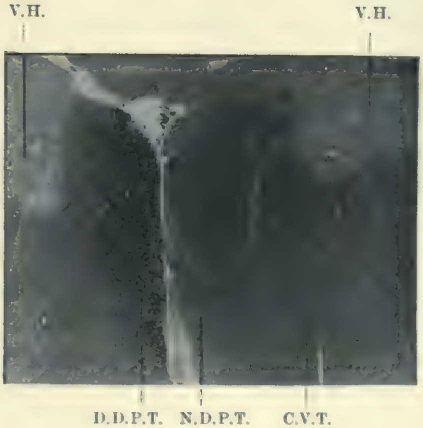


Fig. 12.

throughout the cord, and are still a distinct bundle in the last sacral segment; the degenerate fibres in the crossed pyramidal tract on the side of the lesion (homolateral fibres) can also be traced as far as the last sacral segment, but only a few scattered degenerate fibres are to be seen at this level, while those of the direct pyramidal tract can be traced with certainty to the last sacral segment, and form a distinct though scattered group, chiefly in the dorsal part of the anterior column. As is usual in the case of the crossed pyramidal tract, the degenerated fibres are grouped nearer and nearer to the periphery of the cord, as we pass towards its caudal extremity; but numerous degenerate fibres are seen in the lateral limiting layer through which they probably pass to reach the grey matter; while it is of interest to note that those of the direct pyramidal tract in the ventral region of the cord, on the other hand, leave the periphery and group themselves in the dorsal part of the anterior columns immediately in front of the anterior commissure, as we reach nearer and nearer to the caudal extremity of the cord.

Throughout the sacral cord numerous degenerated fibres are seen passing in the anterior commissure, and are evidently derived from the direct pyramidal tract which is degenerated. These fibres pass from the side of the degenerate direct pyramidal tract to the grey matter of the opposite side. At no other level of the cord have I been able to trace any crossing of these fibres in the way that they are seen to behave throughout the sacral region. In addition to the fibres already described as decussating, there are some which appear to take an opposite course in their passage from the grey matter of one side to that of the other, and are possibly derived from the crossed pyramidal tract.

A Large Cyst of the Centrum Ovale of one Cerebral Hemisphere, the Result of the Degeneration of a Malignant Tumour.

In this case the right cerebral hemisphere was the seat of a large cavity measuring about 7 cm. in its antero-posterior, and 5 cm. in its transverse diameter, occupying the centrum ovale, and extending to a depth sufficient to involve the upper surface of the basal ganglia.

This case like the last proved to be one in which the Marchi method was of the greatest possible advantage for tracing the degenerations. As in the last case, the mode of behaviour of the degenerated efferent fibres in the spinal cord is all that I propose to deal with in the present paper.

In the medulla oblongata, prior to their decussation, the left pyramid is intact, while the right (that on the side of the cerebral lesion) is markedly degenerated, though, of course, to a less pronounced degree than in the other case with a much more extensive cerebral lesion. This case also differs from the last in two other respects, in that no degenerate fibres are seen in the inter-olivary stratum and there is no evidence of degeneration in the tract of fibres at the periphery of the cord opposite to the inferior olive.

At the decussation of the pyramids the degenerated fibres of the pyramid stream off to the opposite crossed pyramidal tract. No degenerated fibres are seen passing to the lateral pyramidal tract on the side of the lesion.

There are no degenerate fibres at the periphery of the medulla opposite the tip of the ventral horn as in the other case, and there are also no degenerate fibres at the dorsal end of the normal pyramid between the grey matter of the ventral horn and the band of degenerate fibres from the pyramid of the side of the lesion, in their passage to the opposite crossed pyramidal tract.

In the cervical cord the degenerated fibres are limited to two regions of its transverse area, the opposite crossed pyramidal tract, and the direct pyramidal tract on the side of the lesion. There are no degenerate fibres in the crossed pyramidal tract of the side of the lesion, or in any tract other than the two just named. The degenerate fibres in the direct pyramidal tract are few and scattered, while those in the opposite crossed pyramidal tract are very much more numerous, and occupy an area much wider in extent than that usually assigned to the crossed pyramidal tract. These degenerated fibres are seen in the greater part of the lateral region of the cord dorsal to the lateral horn, with the exception of a band at the periphery of the cord in the situation of the direct cerebellar and dorsal part of the afferent ventro-lateral tract; but even this area is not quite free as at about the region of the junction of the two afferent tracts, degenerate fibres are seen reaching quite to the periphery of the cord.

The lateral limiting layer is also invaded by degenerate fibres of the crossed pyramidal tract, and ventrally, such degenerate fibres are seen on the outer side of and close to the lateral horn; they extend to just on the ventral side of this horn of the grey matter.

In the thoracic cord degenerate fibres are still seen in both tracts, though fewer than in the cervical region; those of the crossed pyramidal tract are, however, just as scattered as in that part of the cord, occupying an area far greater than that formerly supposed to belong to this tract.

The lumbar cord is characterised by a few degenerate fibres in the dorsal part of the direct tract, in front of the anterior commissure, while an extensive area of degeneration still represents the opposite crossed pyramidal tract. The latter tract is as usual wedge-shaped in this region of the cord, the base of the wedge reaching quite to the periphery, while its apex encroaches on the lateral limiting layer.

A degenerate fibre here and there is all that remains of the degenerated part of the direct pyramidal tract when the first sacral segment is reached, while a fair number of degenerated fibres are seen throughout the whole area of the opposite crossed pyramidal tract at this level; the diminution in the number of such fibres even in this tract is, however, marked.

No degenerated fibres are seen crossing in the anterior commissure from the degenerate direct pyramidal tract to the opposite ventral horn, at any level examined.

Summary of Results.

In brief, then, the facts which appear to be the outcome of these investigations are:—

(1) Fibres derived from the first sacral posterior root while on entering the cord, first occupy that part of the postero-external column which abuts on the grey matter of the posterior horn, subsequently become scattered so as to occupy all parts of the posterior columns, including the cornu-commissural zone and septo-marginal tract, a region supposed to be exclusively occupied by fibres of endogenous origin.

(2) Reflex collaterals derived from this root pass through the grey matter to the anterior horn of the same side, but not beyond it to the white matter of the ventral part of the cord, nor can any fibres be seen decussating to the opposite posterior columns, ventral horns, or other part of the opposite half of the cord. Such fibres are only seen passing to the anterior horn in the segment of the cord to which the root corresponds and that immediately on its cephalic side, viz., the 1st sacral and 5th lumbar segments; no such connection is detectable in other lumbar segments or in the thoracic or cervical regions of the cord.

(3) According to the already well-established law fibres having this origin are limited to the dorsal part of the posterior-internal column in the cervical cord, and are all destined to terminate in the nucleus of the funiculus gracilis.

(4) The band of degeneration in the spinal cord, consequent on destruction of the 7th cervical root, is limited to the postero-internal column, and does not encroach on the limits of the postero-internal column in its course to the cuneate nucleus in the medulla.

(5) The degeneration of this root four segments above its point of entry into the spinal cord occupies the whole dorso-ventral length of the postero-external column, as seen on transverse section, but few of the fibres of the cornu-commissural zone escaping.

(6) The afferent ventro-lateral tract commences to take origin in the spinal cord as far caudally as the third lumbar segment.

(7) In the lumbar, and lowest part of the thoracic region of the cord, this tract is situated at the periphery just ventral to the position of the crossed pyramidal tract.

(8) In the upper cervical cord, on the other hand, the tract occupies an area considerably removed from the periphery of the cord, and separated from it by a well-marked band of healthy fibres. In addition to this, the tract extends much further ventrally than in the thoraco-lumbar cord.

(9) The efferent ventro-lateral tract, on the other hand is situated at the extreme periphery of the ventral part of the cord in its lower cervical and upper thoracic regions. In fact, the fibres which separate the afferent ventro-lateral tract from the periphery of the cord in the cervical region are evidently those of the efferent ventro-lateral tract. This tract preserves its peripheral position throughout the whole length of the part of the cord through which it can be traced. The part of the tract which can be traced farthest towards the caudal end of the cord, by the Weigert-Pal method of staining, being that which abuts on the anterior median fissure.

(10) The degeneration of the comma tract of Schultze, in the 8th cervical segment of the cord after a transverse lesion at the 6th and 7th cervical segments, occupies the greater part of the postero-external column.

(11) After a transverse lesion of the 6th and 7th cervical region of the cord the comma tract can be traced as a distinct band of degeneration, through seven segments of the cord, to the level of the 6th thoracic segment.

(12) Fibres of the crossed pyramidal tract, derived from the opposite cerebral hemisphere, extend throughout the whole length of the spinal cord.

(13) The fibres which degenerate in the crossed pyramidal tract on the side of the lesion (homolateral fibres) pass to this tract from the degenerate pyramid, at the decussation at the caudal end of the medulla, instead of passing to the opposite crossed pyramidal tract in the way that the bulk of the degenerated fibres do.

(14) These homolateral fibres pass to the caudal extremity of the cord, and can be seen with certainty as far as the last sacral segment, where, however, only a few such fibres are to be seen.

(15) The direct pyramidal tract extends throughout the whole length of the cord, in that degenerate fibres can be distinctly seen in the anterior columns as far caudally as the last sacral segment.

(16) Fibres derived from the direct pyramidal tract appear to decussate throughout the whole length of the sacral cord, crossing in the anterior commissure to the grey matter of the opposite side.

(17) Every lesion of the brain, which results in degeneration of the pyramidal fibres, does not of necessity cause degeneration of those of them which pass to the lateral pyramidal tract on the side of the lesion (homolateral fibres), as shown by the case of tumour of the centrum ovale, with degeneration of the direct and opposite crossed pyramidal tracts, without any degenerate homolateral fibres being found.

(18) The real mode of origin of the efferent degenerate fibres seen in the cervical cord, occupying regions other than those belonging to the direct pyramidal tract on the side of the lesion, and to the two crossed pyramidal tracts, is doubtful. The degenerate fibres in the inter-olivary stratum on the side of the degenerate pyramid are the continuations of degenerated fibres which exist in the fillet throughout the pons, and which may possibly be the result of the lesion of the optic thalamus.

(19) All that can at present be said with regard to the efferent tracts, other than those occupying the usual areas of the pyramidal fibres, is, that they are probably composed of internuncial fibres, and that there seems little doubt that the tract seen at the periphery of the medulla, external to the inferior olive, and traced to the cervical cord, where it occupies a limited region at the periphery opposite the tip of the ventral horn, is distinct from the degenerate pyramid, and that its fibres do not appear to be derived from this source.

(20) The degenerate fibres seen in the dorsal part of the otherwise normal pyramid, at the caudal end of the medulla, close to the root of the ventral horn, and between it and the decussating degenerate fibres from the pyramid on the

side of the lesion, may be derived from the degenerate pyramid, or they may be derived from the degenerate fibres seen in the inter-olivary stratum, in that no degenerate fibres from the latter source are seen passing to the posterior column nuclei by way of the internal arcuate fibres; but from which of these sources it is not at present possible to say with certainty. The possibility of their being derived from the degenerated fibres in the inter-olivary stratum is, however, further suggested by the fact that in the case of cyst of the centrum ovale, neither the degenerate fibres in the inter-olivary stratum nor those in the dorsal part of the opposite pyramid are seen. Too much cannot be made of this argument, however, in that in this case not even all the fibres, which we expect to find degenerated in a lesion of the cephalic end of the pyramidal system, are found in this condition, for, as far as can be ascertained, the homolateral fibres in the lateral pyramidal tract on the side of the lesion are intact. Under such circumstances it is possible that other fibres, such as those now under consideration, though derived from the pyramidal system, may escape degeneration in a limited lesion of the cerebrum, in the same way that the homolateral fibres have. But from whichever source these fibres are derived, it is clear that they belong to a decussating system, in that the pyramid in which they are found is absolutely intact on the cephalic side of the pyramidal decussation, unless we assume that they result from compression of the healthy pyramid by the swollen fibres of the degenerated pyramid, in the way that Hallopeau and Rothmann attempt to account for the degeneration of the homolateral fibres.

COMPARISON OF RESULTS WITH PREVIOUSLY ASCERTAINED FACTS.

It will be profitable for us to compare the results that have been just summarised with those facts which have been previously ascertained by similar investigations in man, and by experiment, or otherwise, in animals. In doing so it will conduce to clearness if we preserve the same order of considering the various tracts as has been observed in previous parts of the paper, dealing with the afferent tracts first, and the efferent tracts subsequently.

The Posterior Columns.

Instances in which there has been an opportunity of examining the spinal cord in man after lesions of nerve roots are so rare, and especially instances in which a single root has been damaged, that the two cases here recorded are of considerable value. Moreover, in that the case of degeneration of the first sacral roots allowed of the Marchi method being used to trace the degenerations, it is of special value, as allowing us to trace connections of the collaterals of this root, which would have been impossible by the Weigert-Pal method.

The previously recorded cases of destruction of a limited number of roots in man, in which the degenerations in the spinal cord have been studied, are as follows: A case by Gombault,[8] in which the 5th cervical root was affected; one by Sottas,[12] in which the 6th and 7th cervical roots were involved; another by Souquez,[13] in which the 7th cervical root was completely atrophied; a case by Déjérine and Thomas,[7] in which the 8th cervical root was markedly degenerated, while the 1st thoracic was only slightly so affected; a case by Pfeiffer,[11] in which the 1st and 2nd posterior thoracic roots were affected; and lastly, a case by Nageotte,[10] in which the lesion was limited to the 2nd and 3rd thoracic roots.

It will thus be seen that all the cases of the kind previously recorded deal with the cephalic end of the spinal cord, from the 5th cervical to the 3rd thoracic roots. The case of destruction of the 7th cervical root here recorded belongs to this group of cases, two of which are available for comparing directly with the results arrived at in it, viz., that of Souquez,[13] in which the 7th cervical root was alone atrophied, and that of Sottas,[12] in which the 6th and 7th cervical roots were involved. In both these cases the degeneration in the spinal cord occupied a position in the postero-external column identical with that met with in the present case, and, as in it, the band of degeneration remained limited to the postero-external column, coming up to the septum intermedium without encroaching on the limits of the postero-internal column, and terminating in the cuneate nucleus. This case, therefore, tends to strengthen the previously established fact that the postero-internal column of the cervical cord is composed almost entirely of fibres derived from the thoracic and lumbo-sacral roots, and that few fibres derived from the cervical roots take any part in the formation of this column.

If we next turn to consider the case of degeneration of the 1st sacral roots we find that it is practically unique, and that of the cases of previously recorded limited root destruction in man none are strictly comparable to that now under discussion, for I know of no case of single root destruction in which the affected root has been one of the sacral series. There have, however, been some carefully recorded observations by Souquez and Marienesco, Darkschewitch, and Déjérine and Spiller, of the results of destruction of the *cauda equina* in man. The case observed by Déjérine and Spiller,[6] who were fortunate enough to examine the cord since the Marchi method has been in vogue for tracing degenerated nerve fibres, serves well for the study of the connections of the exogenous fibres of the spinal cord, derived from the lumbo-sacral roots, especially as the cord escaped injury, the nerve roots alone being involved. It may be profitably compared with the results of the single root degeneration met with in the case here recorded.

To discuss the problems connected with the degenerations in these cases properly, it is necessary for us to keep before us the fact that the posterior columns in the lumbo-sacral region of the cord are composed of endogenous as well as exogenous fibres; that is, that in addition to the fibres which pass to these columns from the posterior roots, there are others in it which take their origin in the grey matter of the cord itself. These endogenous fibres occupy two regions of the posterior columns, as seen on transverse section; the one set of fibres that has been alluded to in previous parts of this paper as the cornu-commissural zone, being situated in the ventral part of these columns bordering on the posterior commissure, septum and horn, and the other, whose position varies at different levels, being situated in the part of the dorsal portion of these columns which abuts on the posterior median septum, and which has been previously alluded to as the "septo-marginal tract," adopting the terminology of Bruce and Muir.[16] The former of these tracts of fibres is that to which Marie has applied the term cornu-commissural tract, and is identical with what German writers describe as the "ventrales Hinterstrangsfeld," while the latter is the same as what Obersteiner[21] calls the "dorso-median sacral bundle," and Edinger the "medianes Hinterstrangsfeld," and in one part of its course at the most caudal end of the lumbar region of the cord corresponds closely to the "oval field" of Flechsig. With regard to the variations in the position of the septo-marginal tract, the reader is referred to papers on the subject of endogenous fibres by Dufour,[17] and by Bruce.[15]

Both of these tracts of fibres have been regarded as occupying parts of the posterior columns entirely composed of endogenous fibres, but Déjérine and Spiller[6] found that in their case of compression of the *cauda equina* degenerate fibres of exogenous origin were present in both these areas of the posterior columns, so that they contend that while these tracts are largely composed of endogenous fibres, others of exogenous origin are also met with within their confines. The case of degeneration of the first sacral roots, here recorded, supports their contention, in so far as the cornu-commissural zone is concerned, for the presence of numerous degenerated fibres in this zone, at the 3rd lumbar segment of the cord is unquestionable. It is interesting in this connection to note that this zone, containing so many degenerated fibres in the 3rd lumbar segment, is free from any such degeneration in the 1st sacral and 5th lumbar segments. This case also confirms the observation of Déjérine and Spiller as far as the septo-marginal tract is concerned, in that degenerated fibres have been found in this area of the lumbo-sacral cord in some sections. With degenerated fibres scattered all through other parts of the posterior columns this tract remains free from any such abnormality in some sections, while in others the presence of degenerated fibres in it is distinct.

On further comparing this case with that of Déjérine and Spiller, we find a close agreement as regards the mode of distribution and connections of the reflex collaterals which have been found degenerated.

These observers have, like myself, traced such degenerated fibres to the region of the ganglion cells of the ventral horn on the side on which the degenerated root enters the cord, and our observations are further in accord in that I have failed to find any evidence that such fibres pass beyond the grey matter of the ventral horn to reach the ventro-lateral tracts of the cord, as is supposed to be the case by Edinger, Berdez, Schäfer, and others. Déjérine and Spiller did not find any degeneration sufficiently precise to enable them to support this hypothesis. On the other hand, like myself, they have traced a few collaterals across the base of the posterior horn towards the lateral column of the same side; but I have not seen any such degenerated fibres actually in the lateral columns, nor do these observers say that they have done so. They also describe the degeneration of the fine collaterals in the external part of the gelatinous substance of Rolando, as has already been described in my case. In that their case was one in which all the lumbar roots with the exception of the first pair were degenerated, they were able to trace degenerated collaterals to the column of Clark also, a condition of things that has not been found possible in my case of partial degeneration of the 1st sacral root, in which, as we have already seen, it has not been possible to trace any degenerated reflex collaterals in segments of the cord other than the 1st sacral and 5th lumbar.

My case supplies further evidence of importance as regards the question as to whether any fibres derived from the posterior roots of one side cross to the opposite posterior column. So limited was the degeneration of the root on one side that, as we have seen, no degenerations were met with in the corresponding posterior columns three segments above the point of entry of the root into the cord. This allowed me to make out definitely that no fibres passed from the more degenerated posterior root to the opposite posterior columns.

If we next compare the conditions met with in these cases with what has been found experimentally, we find that there is a close agreement with Mott's[3] results of section of lumbo-sacral posterior roots in the monkey, for the findings of this observer do not confirm the results of Löwenthal,[2] Berdez,[1] Oddi and Rossi,[4] and others who have found extensive degeneration in the white matter elsewhere than the posterior columns of the same side—Mott[3] concludes, from eight observations, that fibres do not pass into the opposite posterior column from divided roots on one side of the cord, and prefers to regard any degeneration met with in the crossed afferent tract of Gowers and Edinger as depending on vascular changes in the grey matter of the posterior horn, rather than by supposing that fibres from the posterior roots pass directly across to ascend the cord and reach the corpora quadrigemina. It will thus be seen that Mott's conclusions are in entire harmony with the facts ascertained in my case of degeneration of the 1st sacral root, where the experimental error of interference with the vascular supply of the cord is naturally excluded.

Before passing to the discussion of the results obtained in connection with tracts other than the posterior columns, the Comma tract of Schultze, contained in these columns, calls for brief consideration. This tract though first noted by Schultze[24] in 1883, was previously figured, though not described, by Bastian[22] in 1867, and Westphal[26] in 1880. Tooth[25] was able to trace it through seven or eight segments of the cord, after a transverse lesion at the level between the 8th cervical and 1st thoracic roots, and through seven segments in a case in which the transverse lesion of the cord was precisely in the same position as in my case, viz.: between the 6th and 7th cervical segments. It is of interest to find a tract, whose extent is admittedly so variable, having precisely the same length of course, after a lesion at the same level of the cord, as was met with in Tooth's case and in mine. This tract is said to be composed of fibres from the posterior roots of the same side which turn back and take a caudal course after entering the cord, and also of other fibres, which pass in the same direction, but take origin in the cells of the cord itself. The researches of Cajal, Lenhossek and Kölliker have proved conclusively that fibres of the posterior roots after entering the cord divide, some taking a cephalic and others a caudal course, but it is curious that after section of posterior roots in animals no evidence of degeneration of the branch which passes caudally has been found. If we relied on the experimental evidence alone, therefore, we should be obliged to conclude that all the fibres of which this tract is composed take their origin in cells in the spinal cord. But in their case of atrophy of the 8th cervical and 1st thoracic roots, Déjérine and Thomas[7] found evidence of degeneration of the branch which runs caudally, as did Nageotte[10] in his case of a lesion of the 2nd and 3rd thoracic roots.

The Direct Cerebellar Tract.

Nothing that we have obtained directly in connection with this tract calls for comment, but in that in the case of the tumour of the caudal extremity of the lumbo-sacral cord the afferent ventro-lateral tract was degenerated on one side, while the direct cerebellar tract escaped on both sides, we have negative evidence of value in confirming the previously ascertained fact that, inasmuch as the direct cerebellar tract probably takes its origin in the cells of Clarke's column, and inasmuch as this column of cells only commences to be at all well marked at the 12th thoracic or 1st lumbar segment, though a few cells may be found as far caudally as the 2nd or 3rd lumbar segments, in a lesion of the cord at the 3rd and 4th lumbar segments, as in our case, it may well escape degeneration.

The Afferent Ventro-lateral Tract.

With regard to this tract, the position of things is different in that we have evidence of a direct kind with regard to it. In the first place, we have seen that if we allow that its degeneration is consequent on the destruction of the cord caused by the tumour directly, then we have evidence that this tract commences as far caudalwards as the 4th lumbar segment, but if we regard the softening and œdema of the one half of the 3rd lumbar segment of the cord sufficient to induce such a degeneration, as it undoubtedly appears to be, then we must regard the tract as commencing at the level of the 3rd lumbar segment. Whichever view we take does not alter the value of the case materially, in that I know of no previously recorded instance in which the origin of this tract from so caudal a level of the cord has been established in man. In Gowers'[28] case in which this tract was degenerated, the crush to the cord was at about the 11th thoracic segment, while Schultze[30] mentions a case in which the whole lumbar cord was crushed by a fracture of the 1st lumbar vertebra. Mott[29] has, however, shown that mesial section of the spinal cord in the monkey, involving the 3rd, 4th and 5th lumbar segments, results in symmetrical degeneration of the afferent ventro-lateral tracts, so that, at any rate in this animal, the tract takes its origin as far caudally as the 3rd lumbar segment, or it may be even at the 4th or 5th.

The other point of interest with regard to this tract, as brought out by the present investigation, is supplied by the case of transverse lesion of the spinal cord, at the level of the 6th and 7th cervical segments. The position of this tract, as seen in the 5th, and all other segments of the cervical cord on the cephalic side of the lesion, is distinct, and leaves no room for question that it is situated more or less mid-way between the periphery of the cord and grey matter of the ventral horn, the band of fibres between this sclerosed area and the periphery of the cord being absolutely intact. Now, in all figures which appear in books dealing with the subject, and which represent this tract, it is placed at the periphery of the cord. Even in the last edition of Foster's "Physiology,"[27] recently published, and containing new diagrams of the positions of the tracts in the spinal cord, this tract is represented as at the periphery of the cord, and external to the efferent ventro-lateral tract, the very reverse of what obtains in the case recorded in this paper. We must, therefore, either regard this case as anomalous, or the majority of the diagrams in our text-books representing the position of this tract as wrong. That the afferent and efferent tracts must overlap greatly is obvious on comparing fig. 5 with fig. 6; but the evidence supplied by the case from which fig. 5 is taken suggests strongly that the major representation of the afferent tract is internal to the main part of the efferent tract. In all the experimental degenerations of the efferent ventro-lateral tract, which I have had an opportunity of studying, the degenerated fibres have invariably occupied the extreme periphery of the antero-lateral region of the cord. So marked has this always been that I was early led to wonder how, even allowing for intermingling, the afferent tract fibres could find room so close to the periphery as they are usually represented; but this is the first case that has enabled me to observe the point in the human cord since my attention was called to it in the cords of animals. I would suggest, therefore, that the relative position of the afferent and efferent ventro-lateral tracts to each other, while there is no doubt considerable overlapping, is such that the efferent tract is situated externally to the afferent, *i.e.*, nearer the periphery of the cord.

The Pyramidal System.

The peculiarities in the behaviour of the pyramidal system fibres noted in my two cases have been also observed by Hoche,[33] in a case published in the *Neurologisches Centralblatt* for November 1, 1897, while my present paper was in process of preparation, and some time after I had discovered these peculiarities in my cases.

In the first place, the degenerated fibres belonging to the crossed pyramidal tract on the side opposite to the cerebral lesion occupies an area of the lateral region of the cord far wider than that formerly ascribed to this tract. It is especially the degenerated fibres bordering on the ventral horn and extending as far as its ventro-external angle, and those occupying the periphery of the cord more or less in the position of the dorsal end of the afferent and efferent ventro-lateral tracts, which call for comment.

Hoche[33] also noted the scarcity of the homolateral fibres in the crossed pyramidal tract on the side of the cerebral lesion, as in one of my cases. In his case, as in mine, and in one recorded by Déjérine and Thomas,[31] the degenerated fibres are seen leaving the pyramid at certain points, a condition of things first observed in 1894, when I found fibres passing from one pyramid to both crossed pyramidal tracts, in a cat[36] with defective development of the central nervous system, in which one pyramid was entirely absent, and by Mellus[34] and Sherrington,[37] who each found fibres passing to both crossed pyramidal tracts from one pyramid after extirpation of portions of the cerebral cortex in monkeys. That there should have been a complete absence of such homolateral fibres in my second case is possibly to be accounted for by the fact that in a less extensive lesion than in the first case, they have escaped, inasmuch as the degeneration of the crossed and direct pyramidal tracts in this second case is only slight compared with that seen in the first. That the difference does not depend on whether or not the basal ganglia are implicated is shown by the fact that both Mellus[34] and Sherrington[37] found that these fibres degenerated after lesions which were limited to the cortex cerebri in the monkey. The proportion of homolateral fibres noted in my cat, and by Mellus and Sherrington in their monkeys, was such as to lead one to expect a much greater number of such fibres in a case with such an extensive cerebral lesion as the first of the two cases now under discussion. Rothmann[36a] has comparatively recently revived Hallopeau's view as to the mode of origin of these degenerate homolateral fibres, supposing that they result from compression of fibres from the normal pyramid by the swollen fibres of the degenerated pyramid at the decussation. Difficult as it is to disprove such a view, there are many considerations which make it improbable that this is the true explanation.

No special comment is called for as regards the behaviour of the direct pyramidal tract on the side of the lesion, in the cephalic end of the spinal cord, either in Hoche's[33] case or in mine; but a special, and hitherto unobserved, or at any rate unrecorded set of fibres in the opposite anterior column demands our close attention. We have in these fibres to deal, undoubtedly, with a crossed tract, unless we accept, as accounting for their occurrence, an explanation like Hallopeau's in regard to the homolateral fibres, for both in Hoche's case and in mine the pyramid on the side opposite the lesion was absolutely intact on the cephalic side of the decussation of the pyramids at the caudal end of the medulla. The only previous observation at all comparable to this is one by Pitres[35] who has shown that both direct pyramidal tracts may be degenerated secondarily to a unilateral lesion. A comparison of my two cases left me, though in doubt, inclined to regard these fibres as possibly derived from the degenerate fibres seen in the inter-olivary stratum in the case in which this crossed tract in the anterior column was found. This was suggested by the fact that in my other case in which no such degeneration in the inter-olivary stratum, immediately dorsal to the degenerated pyramid was found, no such degenerated fibres were seen in the opposite anterior column. On comparing my results with those of Hoche, however, this does not appear to be the true explanation of the source of these fibres, for though he describes this tract at the caudal end of the medulla, and in the cervical cord, he makes no mention of having seen any degenerated fibres in the inter-olivary stratum in his case. All that Hoche states, is that the one pyramid was degenerated, while the other was absolutely intact on the cephalic side of the pyramidal decussation. Assuming that there was no degeneration of the inter-olivary stratum in Hoche's case, we must conclude that this crossed tract of fibres at the dorsal end of the otherwise intact pyramid, at the decussation, and subsequently seen in the territory of the anterior ground bundles on the inner side of the ventral horn, is in reality derived from the degenerated pyramid. This being the case, one cerebral hemisphere is brought into communication with the spinal centres on both sides of the cord by a direct and crossed tract of fibres in the anterior columns, as well as a direct and crossed set of fibres in the lateral columns of the cord, all of which fibres are derived from one pyramid.

There still remains for our consideration one tract of fibres which, so far as I know, has not been hitherto described at all, I allude to the tract which is so definite in the medulla, occupying the ventral periphery of this part of the hind brain, external to the inferior olive, on the same side as, but quite distinct and separated from, the degenerated pyramid. We have seen that it preserves this peripheral position through the caudal end of the medulla, and throughout the cervical region of the spinal cord, though altering its shape at different levels. In that I have not been able to satisfy myself as to the precise point of origin of these fibres, I prefer, at present, to record the existence of such a tract without entering into any speculations as to its significance, or possible functions.

We have already seen that both the new tracts here described can be traced throughout the cervical cord, but are not seen at any point caudal to the 8th cervical segment. It is, however, different as regards the degenerated fibres seen in the direct pyramidal tract on the side of the lesion, and in both crossed pyramidal tracts, which, of course, pass much farther caudally, all of them as we have seen, reaching the last sacral segment of the cord, though only very few of the homolateral fibres are to be seen at this level.

Most authors have regarded the direct pyramidal tract as terminating about the mid-thoracic region of the cord, and, curiously enough, Foster and Sherrington[32] in Part III. of the seventh edition of "Foster's Physiology," recently published, still give this as the level at which this tract terminates. Tooth,[38] however, as long ago as 1889 traced this tract to the 2nd lumbar segment, while Déjérine and Thomas,[31] by the aid of the Marchi method, have been able, in a case of hemiplegia from a lesion of the internal capsule, to trace the crossed pyramidal tract to the 5th sacral segment, and the direct pyramidal, and homolateral degenerated fibres they have traced with certainty to the 4th sacral segment. It will thus be seen that their observations agree closely with what I have found in one of the cases of degeneration of the pyramidal system, the only difference being that I have been able to trace the degenerated fibres of the direct pyramidal tract, and those of the lateral pyramidal tract on the side of the lesion, to one segment of the cord further caudally than in their case. Moreover, they only mention the crossed pyramidal tract fibres as reaching the 5th sacral segment, while in my case they passed beyond this segment to the tip of the cord. This case has, therefore, allowed of my tracing the three sets of pyramidal fibres farther caudally than has previously been done, after any lesion, much less a cerebral one, in that of previously recorded cases, that by Déjérine and Thomas occupied the position which mine does now.

I am not aware of any previously recorded instance in which fibres derived from the direct pyramidal tract have been shown to decussate in the anterior commissure to reach the opposite ventral horn. It has been argued that these fibres must decussate somewhere in the cord, because in cases in which there had been pronounced degeneration of this tract, there has been no paralysis on the same side during life. The case here recorded, however, furnishes the first actual demonstration of the decussation of fibres of the direct pyramidal tract, this decussation taking place, as we have seen, throughout the whole extent of the sacral region of the cord. This is of interest when compared with the surmises of Foster and Sherrington[32] on this point, in that while admitting that the fibres of the direct pyramidal tract must cross in the cord, they regard the ventral white commissure as the path in which they cross, but conclude that only a small part of the commissure can serve this purpose, and that in the lower regions of the cord, where they assume that the direct tract no longer exists, all the fibres of the ventral commissure must have some other functions. It is not a little curious, therefore, that I should have been able to trace the decussation of the direct pyramidal tract fibres in the ventral commissure of a part of the cord where these authors would least expect to find them.

Efferent Ventro-lateral Tract.

I have so recently dealt with the ventro-lateral efferent tracts which exist in the spinal cord, from the experimental stand-point, in this Journal, that I need do no more than remind my readers that the area of sclerosis at the periphery of the ventro-lateral region of the cord, the result of a transverse lesion at the 6th and 7th cervical segments, represents the position of fibres derived from very different sources. One set of these fibres appears to be derived from the superior corpora quadrigemina (Held [42] and Boyce,[40]) while the precise origin of another set of such fibres is in dispute. According to some they are derived directly from the cerebellum (Marchi,[43] Biedl,[39] and Thomas,[46]) while according to others (Ferrier and Turner,[41] Risien Russell,[45] and Mott,[44]) they take their origin in Deiters' nucleus.

There can be little doubt that had the Marchi method been available for tracing the degenerations in the case of fracture dislocation here recorded, it would have been possible to trace this tract much further caudally than has been possible by the Weigert-Pal method of staining, for in experimental lesions of the medulla oblongata in animals the tract can be traced with ease throughout the lumbar cord, by means of the Marchi method.

BIBLIOGRAPHY.*

(A) Posterior Columns.

I. Exogenous Fibres.
  - (*a*) Experimental Section of roots in animals.
  - (*b*) Destruction of roots in man.

II. Endogenous Fibres.
  - (*a*) Cornu-commissural and Septo-marginal tracts.
  - (*b*) Comma tract.

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* A more extensive bibliography will be found in an excellent critical digest by Dr. Alfred W. Campbell, which appeared in the Winter number of this Journal, vol. xx., after my present paper was in print.

I. Exogenous Fibres.

(a) *Experimental Section of nerve roots in animals.*

[1] Berdez, *Rev. Med. de la Suisse Romande*, 1892.
[2] Löwenthal, *Internat. Monatschrift f. Anat. u. Physiol*, 1893, Bd. x.
[3] Mott, Brain, 1893, p. 1.
[4] Oddi and Rossi, *Archives Ital. de Biol.*, xv., 1891.
[5] Singer, "Sitzungsberichte der Wiener Akad," Bd. lxxxiv., abth. iii., 1892.

(b) *Destruction of nerve roots in man.*

[6] Déjérine and Spiller, *Compt. rend. Soc. de Biol.*, 1895, p. 622.
[7] Déjérine and Thomas, *Compt. rend Soc. de Biol.*, Paris, 1896, p. 675.
[8] Gombault, *Bullet. de la Soc. Anat.*, 1891, p. 622.
[9] Gombault and Philippe, *Archiv. de méd. experim.*, 1894, p. 365.
[10] Nageotte, *Rev. Neurol.*, 1895.
[11] Pfeiffer, *Deutsch. Zeit. f. Nervenheilk*, 1891, Bd. i., Heft 5 and 6.
[12] Sottas, *Rev. de. Méd.*, 1893, p. 290.
[13] Souquez, *Compt. rend. Soc. de Biol.*, Paris, 1895, p. 407.

II. Endogenous Fibres.

(a) *Cornu-Commissural and Septo-marginal tracts.*

[14] Barbacci, *Lo Sperimentale*, 1891, vol. ii., p. 386.
[15] Bruce, Brain, 1897, p. 261.
[16] Bruce and Muir, *Ibid.*, 1896, p. 333.
[17] Dufour, *Compt. rend. Soc. de Biol.*, Paris, 1896, p. 449.
[18] Gombault and Philippe, *Arch. de méd. exper.*, 3 May, 1894.
[19] Hoche, *Neurol. Centralbl.*, 1896, p. 155.
[20] Marie, "Lectures on Diseases of the Spinal Cord," pp. 51, 323, 325; *Gaz. des. Hôp.*, 1894; *La Semaine méd.*, 1894.
[21] Obersteiner, "Nervosen Central Organe," p. 263.

(b) *Comma tract.*

[22] Bastian, *Med.-Chir. Trans.*, 1867, p. 499.
[23] Kahler and Pick, *Arch. f. Psych.*, bd. X., 1880, p. 179.
[24] Schultze, *Ibid.*, 1883, p. 375.
[25] Tooth, "Gulstonian Lectures Roy. Coll. of Phys. Lond.," 1889, p. 37.
[26] Westphal, *Arch f. Psych.*, bd. X., 1880, p. 788.

(B.) AFFERENT CEREBELLAR TRACTS.

[27] Foster, "Text Book of Physiol.," 7th edition, 1897, part iii., pp. 942 and 956.
[28] Gowers, "Diagnosis of Diseases of the Spinal Cord," and *Lancet*, June, 1886, p. 1153.
[29] Mott, *loc. cit.*, p. 6.
[30] Schultze, *loc. cit.*, p. 363.

(c) *Pyramidal system.*

[31] Déjérine and Thomas, *Compt. rend. Soc. de Biol.*, Paris, 1896, pp. 157, 159.

[32] FOSTER, *loc. cit.*, p. 962.
[33] HOCHE, *Neurolog. Centralbl.*, 1897, p. 993.
[34] MELLUS, *Proc. Roy. Soc.*, 1894, vol. 55, p. 208.
[35] PITRES, *Arch. de Phys.*, 1884, p. 142.
[36] RISIEN RUSSELL, BRAIN, 1895, p. 37.
[36a] ROTHMANN, *Neurol. Centralbl.*, Nos. 11 and 12, 1896.
[37] SHERRINGTON, *Lancet*, 1894.
[38] TOOTH, *loc. cit.*, p. 33.

(d) *Efferent Antero-lateral Tract.*

[39] BIEDL, *Neurol. Centralbl.*, 1895, p. 434.
[40] BOYCE, *Phil. Trans. Roy. Soc.*, 1895, vol. clxxxvi., B., p. 321.
[41] FERRIER AND TURNER, *Ibid.*, 1894, vol. clxxxv., B. p. 719.
[42] HELD, *Archiv. f. Anat. u. Physiol.*, 1892.
[43] MARCHI, *Riv. Sper. di Frenatria*, 1886, 1887, 1891.
[44] MOTT, BRAIN, 1895, p. 1.
[45] RISIEN, RUSSELL, *Phil. Trans. Roy. Soc.*, 1895, vol. clxxxvi., B., p. 633.
[46] THOMAS "Le Cervelet, étude anatomique, clinique et physiologique," Paris, 1897.

DESCRIPTION OF FIGURES.[1]

[1] The figures are from photomicrographs taken for me by Mr. L. B. Fleming, of Hanwell.

Figures 1, 4, and 7 to 12 are from sections prepared by Marchi's method, and figures 2, 3, 5 and 6 from sections prepared by the Weigert-Pal method.

FIG. 1.—Degenerated fibres of the 1st sacral root entering the cord. The posterior horn, lateral and posterior columns on the same side, and part of the opposite posterior columns are shown. D.M.S. = dorsal median septum. D.R. = degenerated root. D.H. = dorsal or posterior horn. V.H. = part of ventral or anterior horn.

FIG. 2.—Third cervical segment of the spinal cord from a case in which the 7th cervical root was destroyed. D. 7th C. = degeneration the result of destruction of the 7th cervical root. D.C.T. = degenerated direct cerebellar tract.

FIG. 3.—Fourth lumbar segment of the spinal cord. T. = tumour occupying the whole of one half of the cord, and the posterior columns, &c., on the opposite side. V.M.F. = ventral median fissure. V.H. = ventral horn. D.H. = dorsal horn.

FIG. 4.—Eleventh thoracic segment of the spinal cord, from the case of tumour of the cord shown in fig. 3. D.P.C. = degenerated posterior columns. A.V.-L.T. = degenerated afferent ventro-lateral tract.

FIG. 5.—Section from the 4th cervical segment of the spinal cord in a case of transverse lesion at the level of the 6th and 7th cervical segments. A.V.-L.T. = degenerated afferent ventro-lateral tract.

Fig. 6.—Section taken from the 3rd thoracic segment of the same cord as fig. 5. E.V.-L.T. = degenerated efferent ventro-lateral tract. C.T. = degenerated comma tracts.

Fig. 7.—Ventral part of a section of the medulla through the inferior olive in a case in which a large cerebral tumour was removed. O. = Olive. V.L.T. = degenerated ventro-lateral tract. D.P. = degenerated pyramid. N.P. = normal pyramid.

Fig. 8.—Section at level of decussation of pyramids from same case as fig. 7. D.P. = degenerated pyramid crossing to opposite lateral pyramidal tract region. N.P. = Normal pyramid.

Fig. 9.—Section from same level as fig. 8, to show degenerated ventro-lateral tract (V.L.T.). D.P. = degenerated pyramid. N.P. = normal pyramid.

Fig. 10.—Section from same levels as figs. 8 and 9, to show supposed crossed ventral tract (C.V.T.) at dorsal part of otherwise normal pyramid. D.P. = degenerated pyramid. N.P. = normal pyramid.

Fig. 11.—Section from 5th cervical segment in same case as figs. 7 to 10, showing ventral part of cord. N.D.P.T. = normal direct pyramidal tract. D.D.P.T. = degenerated direct pyramidal tract. V.-L.T. = degenerated ventro-lateral tract. V.H. = ventral horn of grey matter.

Fig. 12.—Section from same level as fig. 11, but showing more of the opposite half of the ventral region of the cord. D.D.P.T. = degenerated direct pyramidal tract. N.D.P.T. = normal direct pyramidal tract. C.V.T. = degenerated crossed ventral tract in the region of the anterior ground bundles. V.H. = ventral horns of grey matter.

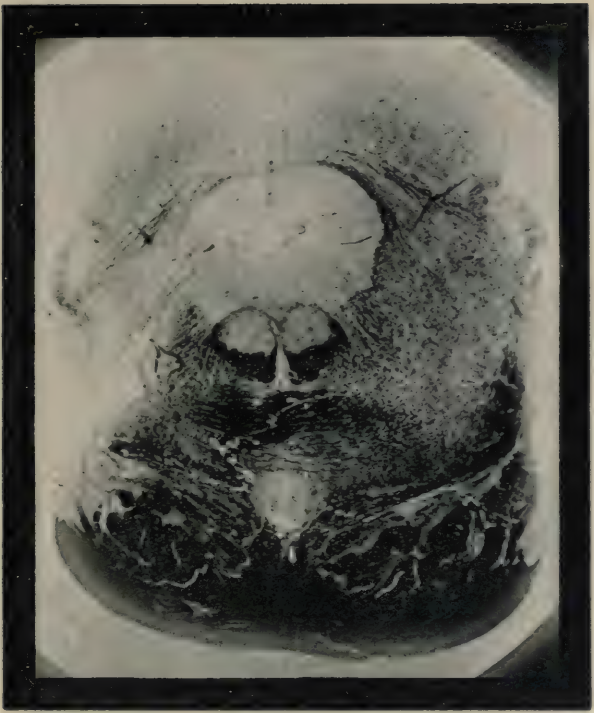
UNILATERAL DESCENDING ATROPHY OF THE FILLET, ARCIFORM FIBRES AND POSTERIOR COLUMN NUCLEI RESULTING FROM AN EXPERIMENTAL LESION IN A MONKEY.

BY F. W. MOTT, M.D., F.R.S.

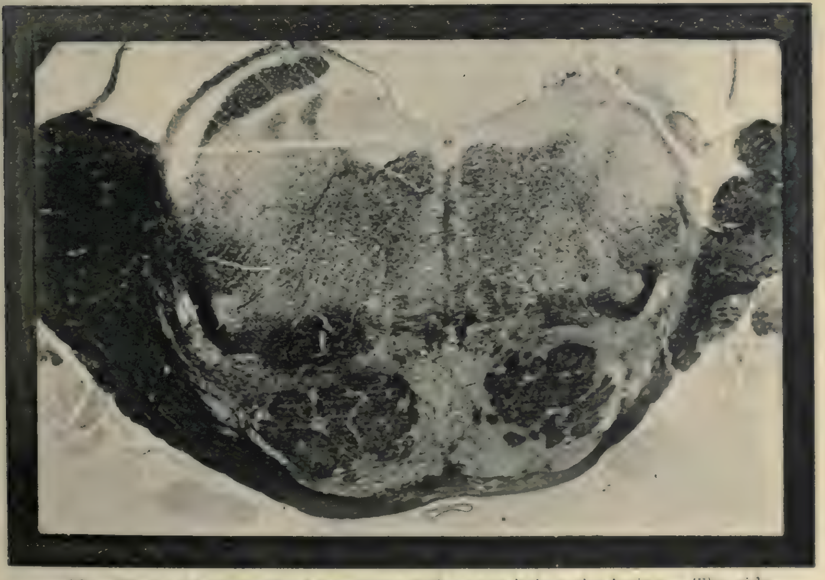
FOUR years ago when making experiments upon the posterior column nuclei (an account of which was published in this journal), I made an attempt to divide the fillet unilaterally on the left side above the region of the pons. The operation was performed upon a young and vigorous callithrix monkey, with a specially constructed knife which was inserted into the cranial cavity through an opening made in the occipito atlantal ligament. When the effects of the anæsthetic had passed off I noticed nystagmus, but it was inadvisable to test it further that day. Subsequently the following notes were made:—

1st day after operation.—Continuous *oscillations* of the eyeballs towards the *left; right hemianopsy* ascertained by the fact that it does not appear to see a piece of apple until it has passed the middle line to the left; discharge from the left nostril. It can move the right arm and leg in associated movements, but not independently. If the finger be placed against the right hand it will not grasp it. A piece of apple is placed upon the left hand (the animal being prevented from seeing it), it tries to look down and seize it; no such attempt is made when the piece of apple touches the right hand. When a clip is fixed on the left hand or left foot it is immediately taken off, even a weak clip; no notice whatever is taken when it is put on the right foot; the animal does not even stop eating, a clear indication that it does not feel on that side. Conjunctival reflexes are present on both sides.

2nd day.—Dr. Hughlings Jackson saw the animal with me, and he considered that there was considerable weakness on the



I.—Photo-micrograph of a section through the seat of the lesion of the fillet on the left side. A scar can be seen extending right through the lateral fillet from the periphery nearly up to the iter.



II.—Photo-micrograph of a section of the pons below the lesion. The side of the lesion is unfortunately reversed, the left side of the pons being on the right side of the plate. Atrophy of the fillet, especially of the medium portion, will be observed, also of the middle and superior cerebellar peduncles. There appears also to be some atrophy of the pyramidal system.

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left side, and in this I quite agreed. Besides the nystagmus and right hemianopsy, one notices most marked rhythmical tremors of the left hand and arm. There is a little power returning in the right arm and leg.

3rd day.—Can now move both legs when struggling; it can grasp with the left foot, but not the right. There is want of co-ordination with the left hand, and rhythmical tremors like those of disseminated sclerosis on attempting to use it. Sensibility on the left side of the body is apparently perfectly normal; whereas on the right side it takes no notice of a clip when fixed upon the arm, leg, trunk, or ear. When touched with a piece of apple on the right side of the mouth it takes no notice, but as soon as the mid line is crossed it bites at it. Knee jerks are exaggerated on both sides.

9th day.—Movement and sensation are both returning on the right side. There is, however, still nystagmus, right hemianopsy, and a tendency to go to the left in progression. Rhythmical tremors of the left hand. Knee jerks exaggerated both sides.

16th day.—Movement and sensation has apparently returned on the right side; it was observed to scratch that side, and it can now hold a piece of carrot in its hand. Nystagmus tremors and hemianopsy as before.

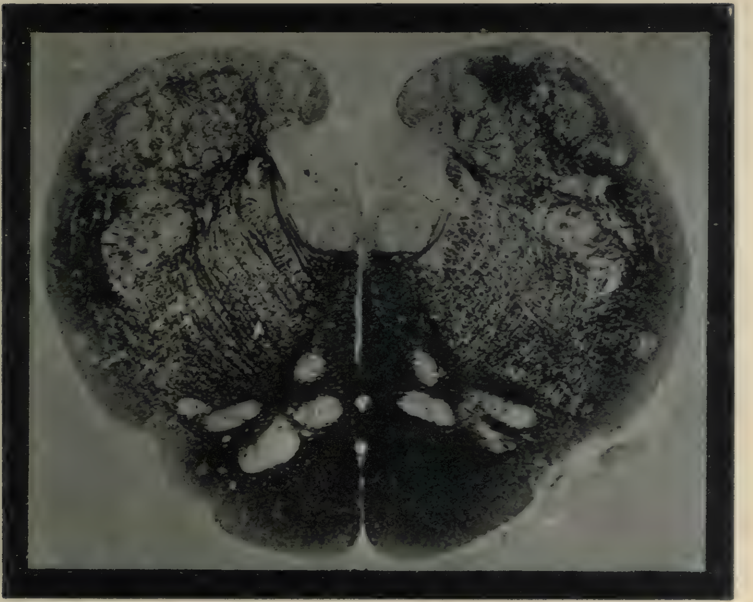
35th day.—Nystagmus still present; tremors less noticeable; still right hemianopsy; sensation is apparently still blunted on the right side, but movements fairly good. The left hemiparesis still manifest. Knee jerks exaggerated both sides.

Gradually these symptoms appeared to pass off, leaving no contracture, and six months later it required a very careful examination to detect any loss of function in the animal. The animal became a pet and possessed great strength and cunning, apparently running and climbing like a normal animal; in only one respect did it show any abnormality in its mode of progression, and that was when placed in an open space it was invariably noticed that it made towards the left, the side upon which its field of vision was unimpaired. It died eventually from pneumonia three years after the operation.

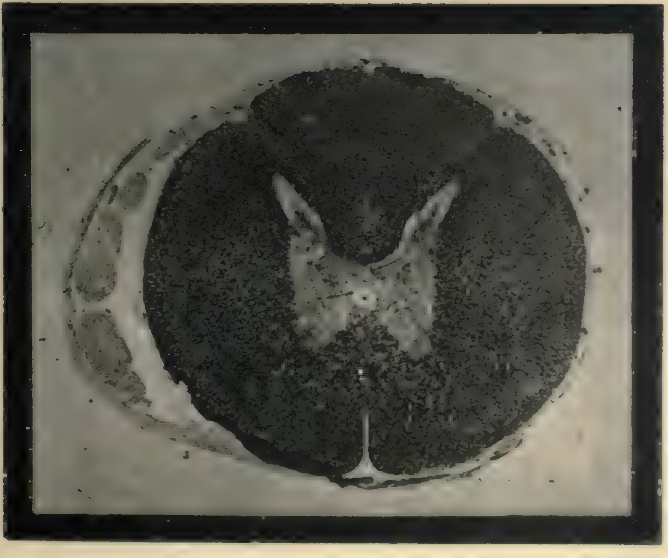
The brain and cord were removed and hardened in Müller's fluid, and sections were cut after embedding in celloidin and then stained by the Weïgert and carmine methods. Serial sections showed that I had injured the optic thalamus slightly, that I had cut through the inferior quadrigeminal body and the external geniculate body; but *the most extensive destruction was in the lateral fillet just above the pons* (*vide* photo-micrograph I.). There was injury of the three cerebellar peduncles and a considerable atrophy of the left lateral lobe of the cerebellum, in fact this was the only naked eye noticeable change on removing the brain.

The lesions were all limited to the left side, and there was but very little scar tissue. I think we may fairly attribute the nystagmus, rhythmical tremors and left hemiparesis to the injury of the cerebellar peduncles; the right hemianopsy to the injury of the external geniculate body; and the hemianæsthesia of the right side to the injury of the fillet and optic thalamus, especially the former. As there is absolutely no sign of degeneration in the crossed pyramidal tracts of the spinal cord, I cannot think that this system was *directly* injured, and we must explain the right hemiplegia by the injury to the fillet and functional disturbance in the motor fibres of the internal capsule, crus and pons in consequence of the lesion.

As the lesion was not a pure one we can only be certain of one or two facts as regards the degenerations that were found. They are—(1) Absence of degenerative sclerosis in the cord, showing that the pyramidal systems were not directly injured. (2) Atrophy of the fillet, especially of the median portion, and atrophy of the corpus trapezoides and inter-olivary layer on the *left side*, with some overgrowth of the glia tissue in these situations (*vide* photo-micrograph II.). (3) Atrophy of the internal arciform fibres and the cells of the posterior column nuclei of the *right side* (*vide* photo-micrograph III.). The fibres and cells are here less numerous and considerably smaller than on the left side. (4) Although Deiter's nucleus was injured I could detect no degeneration in the spinal cord (possibly the injury was insufficient to produce a degeneration extensive enough to be revealed by any other than the Marchi method), (*vide* photo-micrographs IV. and V.). (5) The extensive injuries to the peduncles of the cerebellum of the left side did not produce any evident sclerosis in the spinal cord (*vide* photo-micrograph VI.). The sections were shown at a meeting of the Physiological Society, held at King's College, February



III.—Section of the medulla, showing atrophy of the internal arciform fibres of the right side, and of the inter-olivary layer of the left.



VI.—Section of the spinal cord in the lower dorsal region. There is no apparent sclerosis in the pyramidal system.

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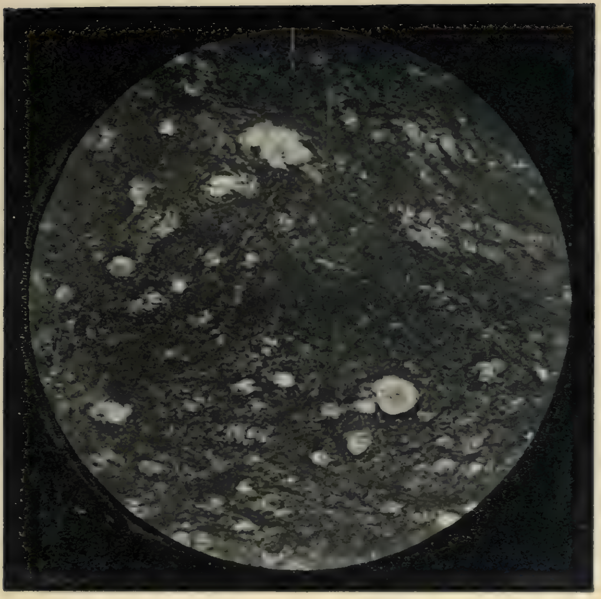
12th, 1898. The accompanying photo-micrographs illustrate the lesion and the descending degeneration and atrophy. Similar appearances of secondary atrophy of the fillet arising from porencephalon have been described in this journal by Alex. Bruce, also by Flechsig and Hösel, Mahaim and Déjérine. Flechsig and Hösel maintain that there is a direct connection between the posterior column nuclei and the central convolutions; that is, that *some* of the axons of the cells of the nuclei of Burdach and Goll pass uninterruptedly to the sensori motor cortex of the Rolandic region, forming what is termed the cortical fillet. Mahaim, Déjérine and Von Monakow maintain that there is not an uninterrupted connection between the posterior column nuclei and the cortex. (6) My observation on monkeys in which I destroyed the posterior column nuclei certainly supported the views of the latter authorities; for in five animals in which I produced most extensive degeneration of the fillet I could find no evidence of degeneration in the internal capsule or cortex. Moreover, the view that the fillet fibres end in the optic thalamus receives strong support from the observations made by Von Monakow, Langley and Grünbaum, Bielchowsky and Jacob, upon material obtained from animals in which extensive *cortical lesions* had been made. They found that the fillet does not degenerate, *but it may atrophy* in consequence of these cortical lesions. I am indebted to Dr. Langley for kindly allowing me to examine his specimens, and I could find no evidence of degeneration of the fillet, although the pyramidal system in the crus on the side of the lesion was entirely destroyed.

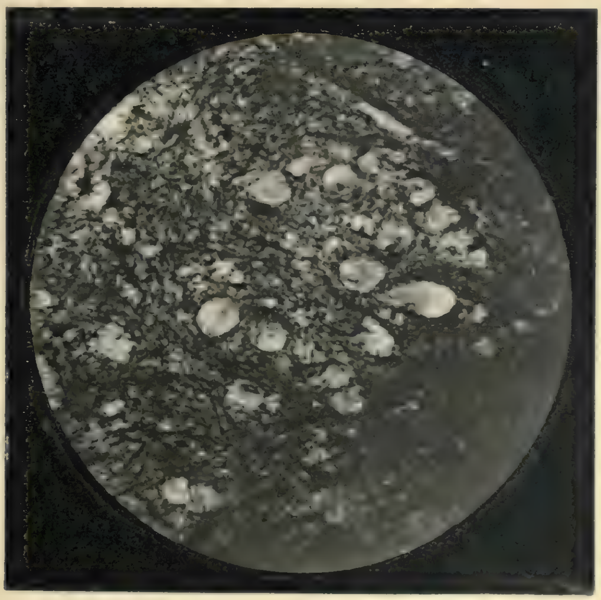
Jacob examined the brains of two dogs operated upon by Professor Goltz. In one there was complete interruption of the fillet in the sub-thalamic region, and a complete disappearance of the fillet fibres below the lesion. In the other the sub-thalamic region was not involved; but there was complete destruction of the cortex, capsule and putamen. In this case there was simple atrophy, but not complete degeneration and absence of fibres in the fillet. He concludes, therefore, that *some fibres do degenerate downwards*, and he puts forward the hypothesis that cells in the optic thalamus give off processes which divide, one branch going to the cortex and the other downward in the fillet. A lesion *below* the thalamus would, however, naturally produce a more marked effect upon the fillet than a lesion above, because if we assume that the fillet fibres all enter the thalamus to come into relation with thalamo-cortical neurons, these latter would have to atrophy and disappear first before the whole of the fillet fibres underwent atrophy.

As a result of the secondary degeneration of the fillet, Von Monakow and Spitzka observed that the arciform fibres of the nuclei of Goll and Burdach undergo a proportional atrophy, and Hösel, Henschen, Bruce and Jacob point out that the cells of these nuclei undergo a secondary atrophy proportional to the degeneration of the fillet. Déjérine believes that in cases of thalamic, subthalamic, or peduncular lesions, there is a retrograde cellulipetal atrophy extending from the periphery of the neuron towards its cell of origin. Bielchowsky examined the brains of two dogs in which Goltz had removed the central cortex and the corpus striatum. The fillet was intact; it showed no sign of descending degeneration. He concludes, with Mahaim and Monakow, that if the thalami are spared, ablation of a hemisphere does not lead in the dog to any secondary degeneration of the fillet. After very extensive cortical lesions involving the whole sensori motor area in monkeys, I have never observed any degeneration in the fillet, although the pyramid was stained black throughout by Marchi method. Redlich has, however, recently described scattered degenerated fibres in the fillet after extensive motor cortical lesions in cats.

The sections from the pons of this monkey, however, show an apparent excess of neuroglia tissue in the interolivary layer of the left side, more, I think, than can be accounted for by condensation due to atrophy of the nerve fibres, so that had I examined the brain by Marchi method, within six months of the operation, I might have found evidence of actual degeneration. The main change is undoubtedly one of *secondary disuse atrophy;* but Jacob's observations suggest that there may have been some true



IV.—Section of the posterior column nuclei to show cells in Burdach's nucleus on the side opposite the lesion. A portion of the section was photographed, which showed the largest cells. *Magnification* 200.



V.—Section of the posterior column nuclei, showing normal cells in Burdach's nucleus on the same side as the lesion. The cells are on the whole much larger than those in the atrophied nucleus. *Magnification* 200.

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descending degeneration in the fillet, as well as disuse atrophy.

As I injured the thalamus, the examination of the internal capsule would have afforded no positive evidence whether there is in the monkey a direct cortical fillet or not. Moreover, no method other than that of Marchi would have been suitable for showing a slight degeneration.

LITERATURE.

BRUCE, ALEX. "A case of descending degeneration of the lemniscus, consequent on a lesion of the cerebrum," BRAIN, 1893.

FLECHSIG AND HÖSEL. "Die Central windungen ein Central organ der Hinterstränge," *Neurol. Centralblatt*, 1890.

MAHAIM, ALB. "Ein Fall von Sekundärer Erkrankung des Thalamus Opt. und der Regio Subth.," *Archiv. f. Psych.*, xxv.

MONAKOW, C. VON. "Allgem. Anat. des Gehirns Ergeb. der Allg. Path. und Path. Anat. des Menschen und der Thieren."

LANGLEY AND GRÜNBAUM. "On the degeneration resulting from removal of the cerebral cortex and corpora striata in the dog," *Journal of Physiology*, 1890.

SPITZKA, E. C. "A contribution to the localisation of focal lesions in the pons and oblongata," *Journal of Nervous and Mental Diseases*, 1890.

DÉJÉRINE, M. ET MME. "Sur les connexions du ruban de Reil avec la corticalité cérébrale," *Compt. Rend. de la Soc. de Biol.*, 1895.

BIELCHOWSKY. "Obere Schleife und Hirnrinde," *Neurol. Centralblatt*, 1895.

JACOB, CHR. "Ein Beitrag zur lehre vom Schleifenverlauf," *Neurol. Centralblatt*, 1895.

REDLICH. "Exstirpation motorischer Rindencentren bei Katzen," *Neurol. Centralblatt*, 1897.

THE CEREBRAL CORTICAL CELL UNDER THE INFLUENCE OF POISONOUS DOSES OF POTASSII BROMIDUM.

BY HAMILTON K. WRIGHT, M.D., C.M.

Medical Registrar and Neuro-Pathologist, Royal Victoria Hospital,
Montreal, and
John Lucas Walker, Exhibitioner of Cambridge University, England.

A SEARCH through medical literature has failed in bringing to light any account of the changes induced in cerebral cortical nerve-cells or their appendages, of either man or lower animals, by the action of either medicinal or poisonous doses of potassii bromidum.

Not long since I obtained the brain of a man who for three years was subject to attacks of idiopathic epilepsy of varying frequency, and who, in error, for a period of eighteen days, consumed continuous excessive doses of the above salt which indirectly, if not directly, seemed to be the cause of his death.

This appeared to be an opportunity for an investigation, and if possible, a determination of any alterations in the cerebral psychical cells, whose functions were certainly disturbed, as will be seen by the clinical history of the case.

But being aware, however, of the fact, that seldom a single isolated example of any morbid tissue metamorphosis can be taken to firmly establish it as a pathological entity, and also of the frailty of any inference drawn, or conclusions arrived at, from the observations of such a solitary instance, I have taken the precaution to control my study by examinations of the cerebra of four rabbits, which were subjected daily to large doses of potassii bromidum for a period of nearly a month. And to make assurance doubly sure I kept two rabbits under identical environment and feeding, without, however, administering any drugs to them.

Moreover, it must be readily seen that in such a case as this, with its antecedent history of psycho-motor disturbance, adequate control and verification by experiment were absolutely necessary to avoid confounding a possible underlying patho-anatomy of the epilepsy itself, with a more recent destructive change produced by the action of the drug.

Clinical History.

Before proceeding to detail the observed modifications in the nerve-tissue of the various cerebra, it will be well to give the history of the patient as learned from his own account, and from the physician in charge. For this history will establish the fact that the case is one of extreme bromidism, and will militate against the assumption that the alterations are merely those of cell exhaustion which without doubt supervene in epilepsy, in consequence of the continuous explosive discharge in the psycho-motor region of the cortex cerebri.

Briefly, the history is as follows:—

The patient, a man aged 26 years, came under observation on February 11, 1897, with a history of *grand mal* of three years' standing.

During the first year the fits occurred once or twice a week, then about once a month for eighteen months, and for six months before coming under notice they attacked him not oftener than monthly. During these last months, however, there were intercalated between the major attacks several fits of *petit mal* every twenty-four hours.

No mental disability attended the onset, or followed the minor epilepsy, but after each major fit there was intense headache and a stupor lasting generally twenty-four hours.

Observation showed him to be slightly neurotic in temperament, and to be subject to frequent attacks of petit mal during each day. Reflex and muscular power appeared to be normal.

No major fits occurred during the time the patient was under observation, but nevertheless he was put under bromide treatment for the minor attacks and for the headache which invariably followed them.

On the second or third day of treatment it was noticed that he became heavy and stupid, and this stupor became more and more marked until death two weeks later. About a week before the latter event, on February 23, muscular weakness supervened, and the man was almost unable to walk or handle the ordinary utensils used in eating; when he did move about it was more as an automatum than as an intelligent conscious being.

On February 27, sixteen days after the commencement of the administration of the bromide, it was observed that he had considerable difficulty in swallowing. On the 28th the difficulty became pronounced and in addition he now failed to recognise his friends; urine was passed involuntarily. After March 1 no food or liquid was taken by the mouth, as the patient had several attacks of choking that left him extremely cyanosed. Also there developed on this day a few moist râles in the lungs. He became comatose and his lower jaw relaxed. Brandy and milk were given through a stomach tube in order to sustain life. Respirations now became rapid, thirty-six per minute, and the temperature rose to 100·25° F.

On March 2 his tongue became swollen and the administration of stimulants and food had to be discontinued because of the choking produced by the passage of the gastric tube. On the afternoon of this day the temperature rose to 104·1° F., the respirations to 48 per minute, and the pulse rate increased to 132.

Next day, March 3, bilateral bronchitis became pronounced, and before evening this had developed into a pneumonia affecting both sides. In the evening his eyes were examined and an intense bilateral optic neuritis was found to be present, and the double conjunctivitis which had set in a week before became more marked. In addition there was an abrasion of the corneal epithelium on both sides. At 5.30 p.m. of this day the pulse rose to 240 and maintained this or a higher rate until death. The temperature gradually heightened from that hour on until it reached 108·4° F., just prior to death at 3.30 a.m of March 4.

From the time the patient came under observation on February 11 until interference with deglutition occurred on the 29th, he took ostensibly twenty grains of potassii bromidum three times a day, but really he had consumed 160 grains three times per diem, thus making a whole ounce in twenty-four hours. The error arose through the solution of the salt being 1 in 3; and the patient had been given an ounce of the mixture three times daily, instead of a drachm diluted to an ounce with water, as was directed by the physician in charge.

It will be seen, therefore, that the quantity of the drug taken by the patient was great, and quite sufficient to cause the onset and persistence of the motor and psychic phenomena of extreme bromidism such as occurred on, and continued from, the third day of the administration of the salt.

Careful analysis of the above record of the patient will show that, in no period of his illness was he subject to a large number of rapidly recurring convulsive outbreaks, that for the six months before the commencement of the bromide administration he had but one major fit a month. and that during the eighteen days in the hospital only *petit mal* with subsequent headache was noticed.

This history, together with the fact that competent observers are not agreed as to the presence of morbid changes in the cerebral cortical cells of those who have suffered from idiopathic epilepsy, even when most carefully examined by approved modern methods, justifies my assumption that any changes exhibited by the sensori-motor cells of the cerebrum under discussion may be regarded as certainly not attributable to the primary cause of the epileptic manifestations themselves, nor to any condition induced as a result of such manifestations. They must, therefore, be ascribed to the effect (1) of poisonous doses of potassii bromidum, or (2) to too long an exposure after death of the brain before it was immersed in a fixing medium, or (3) possibly to the action of the fixing medium.

The necropsy was performed twelve hours after death, but during this lapse of time the body was kept at a temperature of between 20° and 30° F. in the mortuary refrigerator of the laboratory, and at the time of the examination presented no sign of decomposition in any of its parts. Yet without doubt it must be allowed that some *post-mortem* alteration took place, some fine change, even though not obvious, and this will have to be considered in a final summing up of the case.

MACROSCOPIC OBSERVATION OF THE HUMAN BRAIN.

Naked eye examination of the human brain disclosed the following: slight œdematous effusion over the whole cerebral cortex, and a noticeable capillary distension in the frontal and parietal pia mater; the lateral ventricles contained the normal quantity of fluid and their ependyma was healthy. No spots of softening or induration could be felt in the cortex that would point to the possibility of the convulsions being of organic cause.

Without being cut into by any of the generally used laboratory methods farther than was just necessary to expose the lateral ventricles, and with only a partial removal of the pia-arachnoid, the entire brain was placed in a 3 per cent. solution of formaline, and this was repeatedly renewed until the tenth day, when the brain was removed and divided by a sagittal cut into right and left hemisphere, and then each half by transverse-cuts into slices about two cm. in thickness. No gross alterations of any kind were to be seen in the cortex, medullary centre, or internal ganglia, and the tissue was re-immersed for more complete fixation. At the end of another week the brain was well hardened throughout and fit for microscopical investigation.

Small pieces were then taken from the superior, middle, and inferior aspects of the pre-central and post-central convolutions of each hemisphere, corresponding roughly to the lower and upper limb and face centres respectively; and also from the frontal and occipital lobes. But as the examination of these showed no differences according to position, the situation from which the pieces were taken may be entirely disregarded.

After being thoroughly washed, the pieces were halved and a moiety of each dehydrated in alcohol, carried through celloidin, and an attempt made to stain the sections therefrom by hæmatoxylin-eosin, Nissl's, and Unna's polychrome dyes. The remaining halves were treated to a week's bath in Müller's fluid, as they were intended for examination by the silver method.

It is my experience that nerve cells and their fine constituents are inclined to stain badly with aniline dyes if the fixing medium had been only formaline. [The case under consideration is no exception to the rule, and I have, I regret to say, to confess to somewhat meagre results from their use in this case.]

Still with Nissl's fuschin method, and by the use of Unna's polychrome, changes are shown in the cells which tell of the deleterious action of some toxic agent, and which supplement the alterations made out by the silver stain used in the case. They also corroborate the demonstration of changes in the blood-vascular tissues afforded by the hæmatoxylin-eosin method.

Minute Anatomical Changes.

Blood-Vascular Alterations.

The pial vessels are dilated wherever observed and are filled with corpuscles, and so also are the various sized vessels that pass from the surface into the cortex and medullary centre. Careful examination, however, discloses no gross injuries of the vessel walls, and no noteworthy increase in number, or alteration in quality of the erythrocytes or other blood-elements. Yet a fine lesion is to be observed in the vessel wall which consists of a slight swelling and inwards protrusion of the endothelial nuclei and a more marked avidity manifested by them for the various dyes.

The larger vessels are not the seat of any considerable change. But the nuclei of the endothelium are swollen and bulge well into the lumen of the canal.

In the medium sized vessels the turgescence of the endothelial nuclei is the salient and only change; if anything it is more pronounced than in the larger vessels. In some instances the nuclei project inwards so far as to nearly block the tube; but no bulging of the vessel walls on either side of them is to be observed, nor any evidence of stasis in the blood stream from the narrowing of the lumen.

In the capillaries most nuclei are turgescent and deeply stained. Occasionally a slight bulging outwards into the peri-capillary space is noticeable, corresponding to the position of the enlarged nuclei. At no point, however, do the latter impinge so far on the capillary canal as to cause stasis in the blood-stream. The peri-vascular spaces contain a fine granular débris and often polynuclear corpuscles.

It will be seen, therefore, that the blood-vascular tissues have not been acted upon to any great extent by the bromide. The enlargement of the nuclei of the endothelium, the deep staining, the distension and engorgement of the vessels, and the filling of the peri-vascular lymph spaces with cell-detritus are the only manifestations of its action.

Pathological Histology of the Human Cerebral Cortical Cells as shown by the Aniline Stains.

The most salient alteration is a near approach to absolute homogeneousness in the protoplasm of the bodies of the cells. No lamina of the cortex is wholly exempt. As this feature of degeneration, however, appears most evident in the pyramidal strata, cells from these will stand as examples for description throughout this paper.

The amorphous character of the extra-nuclear protoplasm is made evident by both of the Nissl stains, and equally so by polychrome stain. It may be looked upon, therefore, as essentially due to changes of the cell plasm, and not as a consequence of any unknown action of the staining fluids themselves. Many cells, about one in every ten, show absolutely no chromophile particles in their body protoplasm, while others, and these in great number, contain but a few of them irregularly scattered and scarcely distinguishable from the surrounding hyaloplasm. There appears to be no constancy in the point of distribution of the chromophile particles in the cell body. In one a few grains may lie close to the nuclear ring, with other few near the apex or scattered along the basal margin, or the particles may be present in only one of these positions.

In every instance where the complexion of the cell protoplasm has undergone this transformation it is accompanied by evidences of change in the nuclei. It appears to be a rule that in some measure the one shall be attended by the other.

It is to be expected that the nuclear outline should be somewhat hazy and ill-defined when the marked alteration in the encompassing body-plasm is considered. Carefully prepared sections not unusually show almost a blending of the outer margin of the nuclear ring with the body plasm. The inner margin also is ill-defined, and may present a finely dentate contour, the long processes of which proceed inward for a short distance and merge, or end, in the deeply stained nuclear fluid. At first I thought that the dentate and rayed inner margin of the nuclear ring was due to the diffusion of the stain which sometimes occurs. But clean alcohol and aniline oil gave a similar picture, and therefore it is most likely that the condition is a morbid one. The membrane again may not be equally stained throughout, but may have sections of pale, granular material interposed between other sections deeply stained and not in any way altered from the normal.

Often nuclei are seen dislocated from the axial position which they occupy in the normal cells. Instead of lying centrally as in health the nucleus is now displaced, it may be slightly, but in no rare number of cases to the periphery of the cell. No example presented itself, however, of even a commencing extrusion of nuclei from the cells which contain them, providing that the latter are not more degenerate than already described.

The nuclear achromatin in the great majority of all cerebral cortical cells no longer deserves the name, for it has not decolourised in any of the stains. In consequence of this the general appearance of the nuclear vesicle is more homogeneous than in sound cells, and there is a decided lack of refractile brilliance in it.

Morbid changes, however, are not wholly confined to the nuclear membrane and its contained fluid. The nucleolus in most cases is turgescent and blanched, or in a fair way towards complete disintegration. The contour of health is lost, and the smooth roundness is now represented by a granular delicately crenated rim. A still more advanced stage of atrophic metamorphosis is observable in a rarer number of nucleoli. They may be swollen to nearly twice their healthy size and present an etiolated form with a lightly but distinctly dyed margin suggestive of a membrane. The latter is generally round, but may be flattened as though erosion had taken place. On the whole this state of the nucleus is rare. More often there is an irregular staining of its structure. From the inner aspect of its margin delicate rays project, and after crossing about a third of the diameter of the swollen nucleolus they become lost in the more pale central portion.

Are these spurs made up of aggregations of minute dust particles which now overlie the nucleolus, or are they a part of the latter structure itself which has yet retained a sufficient affinity for the dye to successfully resist the action of the decolourant? In view of the fact that they show, even in the thinnest sections, and also that there is a positive alteration in the chemical properties of the rest of the nucleolus manifested by its blanched, turgescent condition, it seems likely that they are a part of the nucleolus not yet wholly implicated in the chemical change which has affected the greater part of the structure. Moreover, similar features obtain in the nucleoli of the poisoned rabbits, and these confirm the conclusion that this feature of degeneration in the human nucleoli is an actual atrophic change and not due to any fortuitous collection of nuclear dust particles.

Other nucleoli show centrifugal blunt spurs, very short, or longer and more sharply pointed which project from the surface outwards into the encompassing nuclear matrix. These are not rare; but I have in no instance been able to follow a ray to the periphery of the nuclear membrane except where a nucleolus almost abuts upon the latter, and then only from that sector of it which faces the nuclear rim. In all these cases of nucleolar metamorphosis the body of the structure is increased in any transverse diameter but is less deeply dyed than in health. In addition, the edges of the shorter spurs may be finely granular.

But the great majority of all nucleoli show no such remarkable morbid features, the change which they present being of milder degree. Most common is a swollen, blanched condition throughout, which may, or may not be accompanied by some aberrancy in form. Yet, be the structure round, oval or flattened, its edges are invariably finely granular throughout, or show in some limited extent a very delicate crenation.

In cells whose nucleoli are in an advanced stage of metamorphosis, or are entirely absent, there are to be seen large angular particles lying in the altered achromatin of the nuclei. These are never so small as the large molecular particles of healthy nuclei. The foreign forms stain deeply and are sharply defined from the abnormally tinted protoplasm in which they are scattered. In form and avidity for stain they closely approximate the large angular particles described by Berkley in the nuclei of the cerebral cortical cells of rabbits that he poisoned by alcohol. More than six were never observed in any one nuclear sack; three or four is the usual number. Never do they approach in size or form the large corpuscular bodies seen and pictured by the above mentioned observer in his experiments with ricin and other equally virulent poisonings. I looked very carefully for these, but neither here nor in the rabbits' cerebra was I able to detect them.

In sections, purposely over-stained, an excellent exhibition is made of the not infrequent enlargement of the main apical processes of the cell bodies. This feature is usually a part of a general enlargement of the entire cell. The basal processes of such cells and their proximal branches are also implicated to some degree in the swelling of the cell body. Slight bulging and dentation is observable in the margins of the processes close to the cells from which they originate. Where enlargement of the cell bodies and their processes is extreme the pericellular spaces are either obliterated or narrowed almost beyond recognition.

Besides the above noted alterations in the cell body there is to be observed a more grave and advanced atrophy wide spread through all cortical layers. Sharply defined and regular contour now no longer obtains. A slight crenation of one side of a cell foreshadows the extreme atrophy that has occurred in another not far removed from it. The crenation may be extreme and affect the whole outline of the cell body, or there may be more than mere crenation. The cell may be puckered or shrivelled, and its protoplasm reduced to an irregularly thin amorphous layer which closely embraces a shrunken homogeneous nucleus. Other cells are ragged in outline with alternate rough projections and deep or shallow bays with frayed margins. In the latter lies a granular débris which is more deeply stained than the cell protoplasm from which it has not improbably been shed. Rarely extreme examples of this are to be seen in which the nuclei are eroded in that segment which is in contact with the disintegrating sector of the cell rim.

The process of disintegration in some instances has not stopped short of almost total destruction of the cell body. In the most remarkable examples the peri-cellular space now contains in its median or lateral aspect a mass of deeply stained granular débris. From the edge of this a fine strand or two, very pale, or irregularly stained, may pass to the margin of the extra-cellular space. Doubtless these are the remnants of once healthy cell processes.

The above described morbid change is certainly one of advanced degree and not far removed from a final disintegration of the whole cell and its appendages. Whether or not many cells have actually split up and been removed by the lymph stream is hard to determine. I looked carefully for absolutely empty cell spaces and found none. In every one that caught my eye was a cell, normal, or in one of the grades of degeneration already mentioned.

Turning now from the proper cortical cells to those whose function is mainly one of support, it may be seen that they are implicated in a slighter degree. A few of the neuroglia nodal points have swollen to twice their normal dimensions, and are pale and granular. Only one or two deeply stained molecules are visible in such. A considerable number is shrivelled up into some aberrant form. They may be kidney, or pear-shaped, and crinkled in part, or in their entire margin. Deeply stained throughout they exhibit no interior molecules and contrast remarkably with the normal nodes in their neighbourhood.

Both types of degeneration prevail almost wholly in the grey cortex, only rarely can an altered glia-cell be observed in the subjacent medullary substance. In the deeper parts of the white matter the support cells are all regular in outline, round, and both their chromatic and achromatic substance is in a perfectly sound state. This is of course what might be expected from the known action of potassii bromidum on the central nervous system, and is certainly a strong point in favour of the change which has affected the proper cortical cells being the result of the action of the excessive doses of the salt given in mistake in this case.

Histological Appearances by Berkley's Silver-Phospho-molybdate Method.

In comprehensive and trustworthy research on the nervous system it is necessary to supplement one method by others.

Berkley has recently given us a method which is apparently adequate as a means of demonstrating the morbid changes in the protoplasmic extensions of nerve cells. Using this mode as described in the 1895 winter number of BRAIN, I obtained good results. Golgi's, and Cajal's methods had been tried upon some of those portions of cortex which had been hardened in Müller's fluid, but with unsatisfactory results.

But cursory examination of the proper cortical tissue successfully stained by the silver phospho-molybdate shows that a severe process has been in operation. Destruction of cell bodies and their appendages is as extensive as that shown by the aniline stains.

Breaches of continuity have happened widely in both axons and neurodendrons, and their alignment is now indicated by globular or club-shaped detritus, or else by well preserved segments. This phase in the cells is, in part, an artefact. For, unfortunately, the tissue was overhardened in Müller's fluid and thus rendered somewhat brittle. The microtome knife, therefore, broke through portions of the dendritic processes and their buds. These, however, generally lie close to their parent cell. Yet in some instances they have disappeared altogether, probably washed away in the process of clarifying and mounting.

It is therefore not easy to determine just where the pathological process ends and the accidental changes begin. So that we must guard against interpreting purely manipulative alterations as changes caused by the poisonous action of the potassii bromidum. Therefore, I have controlled the investigation by observations on other human cerebra that had suffered exposure to the same laboratory conditions. A further difficulty is presented by our ignorance of the action of nitrate of silver on adult nerve tissue which has been altered by disease or *post-mortem* exposure.

On the whole, silver has not yet proved itself to be the ideal stain for the investigation of neuro-pathological changes.

Yet since the attempt first made by Kronthal[1] to use the chrome-silver, many other competent workers have entered the field and have overcome in some degree the difficulties met with in the use of the Golgi method when applied to the investigation of suspected morbid alterations in adult cerebra.

Greppin,[2] in 1893, Colela,[2] and Golgi[4] himself in 1894, and later in the same year Klippel,[5] Azoulay[6] and Andriezen,[7] gave us results obtained by them. All of them also offered them with more or less apology for the possible imperfection of the chrome-silver method.

In 1895, Monti[8] of Pavia, in an experimental research into the effects on canine cortical cells of artificial embolism, achieved happier results.

But it was not until Berkley,[9] of Baltimore, carried out by his improved Golgi method, his series of investigations on artificially induced lesions in the cerebral cortical, and other brain cells of rabbits, that we had a trustworthy demonstration of degeneration of cell appendages by the silver method.

By the use of his modification of the silver method, I find the following departures from the normal which were foreshadowed by the Nissl and polychrome stains.

In all cortical laminæ are cells which are in some stage of degeneration. No stratum appears to have escaped, and the atrophied cells in any one layer agree so closely in number with those in any other, that any difference may be passed over. The change is likewise similar in all, although it seems that the pyramidal cells are most deeply implicated. Because of this and the individual importance of the pyramidal cells attention will in the main be confined to them.

Fig. 1 is a good example of the least degree of change observable in the cortical cells. The cell body is irregular in outline, slightly enlarged when compared with the healthy pyramidal cells, and presents in some part of its rim, blunt cusps which alternate with deep or shallow concavities.

If the primal ascending dendron is followed to its visible termination, several ampullous or varicose swellings of varying size are met with, separated from each other by longer or shorter stretches, not different in form from health except that a great many lateral buds have been shed, thus leaving the stem bared for short distances. The thrown off buds, however, lie on the surface of the section not far removed from the bared margin. Their dislodgement is without doubt manipulative. Quite different from this, is the denudation presented by the before-mentioned varicosities; these are clearly defined in outline and absolutely budless. The tissue in their neighbourhood, except rarely, contains no signs of shed gemmulæ and these are probably a few of those already mentioned as thrown off, but in this case more widely separated from, the healthy portion of the process.

On the basal processes also varicosities are to be seen, but they are small, and like those on the ascending protoplasmic process, are sharp in outline and shorn of the lateral projections which obtain on the unaltered part of the extensions. One may be seen on each secondary branch and ranges in size from a small and scarcely recognisable to a readily obvious swelling. None of them, however, reach the dimensions of the swellings on the apical projection and its branches.

On the latter parts the varicosities follow each other in more or less rapid succession, eliptical, round or oval, and all of about the same transverse diameter. The visible tips of the off-shoots from the main stem may end in an oval

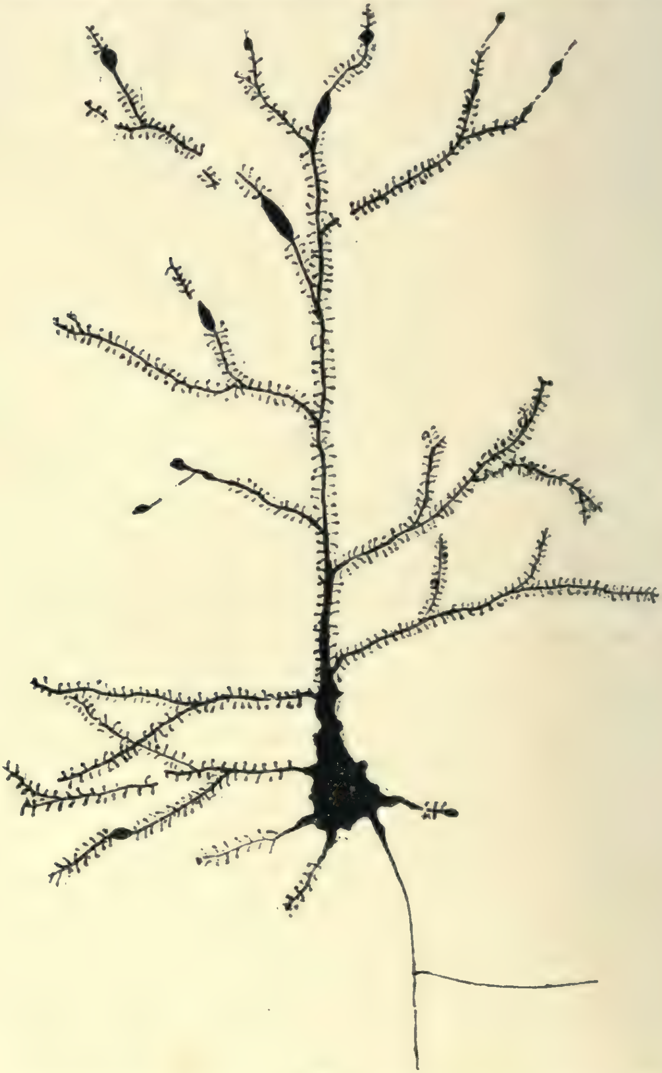

FIG. 1.

Cell body and extensions from the chief pyramidal layer of the human brain. Body covered with projections. Swellings on the dendrons. The break in continuity is artificial.

or sack-like enlargement of great relative magnitude, but generally, however, they are smaller than those observed on the main apical process.

Where branches are given off from the secondary apical processes, and at their splitting point into tertiary ramifications, enlargements are also to be seen; but these are generally forked in shape, thus conforming to the bifurcation of the stem, and in most instances have retained the mossiness of the healthy part of the stem. It is hardly likely, therefore, that these swellings are similar in origin to the first-mentioned, which are denuded of buds.

The axis cylinders of the cells not more deeply implicated than those described are intact so far as they can be traced. Unfortunately, this is not for any great distance, because in most sections they are broken off and carried away.

Cells in the state described above are on the whole not great in number compared with those showing advanced deterioration.

Fig. 3 shows to what an extreme condition many of the cells have been brought by the action of the poison. The cell bodies are irregular in their outline, not infrequently deeply excavated in some one aspect, and showing more ragged cusps than less involved cells. Where processes exist (and the most persistent are the apical), they are swollen, and coarse close to the cell, and farther out are the seat of bulbous enlargements. In this stage of atrophy, however, the latter are larger and more numerous on the basal extensions than on similar parts of the previously mentioned cells. A pathological fragmentation has occurred here, and the globular or granular remains of the smaller pieces yet remain to indicate that the processes were sundered by a morbid rather than manipulative process.

In such cells the various processes are swollen close to the cell body, and after a short course into the sustentacular tissue they end in an oval or round bare swelling. But the ending may not be so abrupt. A gradual dwindling is obvious as the course of the protoplasmic process is followed outward and the ultimate termination may be sharp. Beyond many of such terminations are small, or larger,

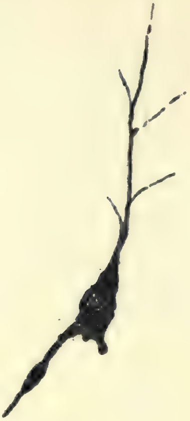

FIG. 2.

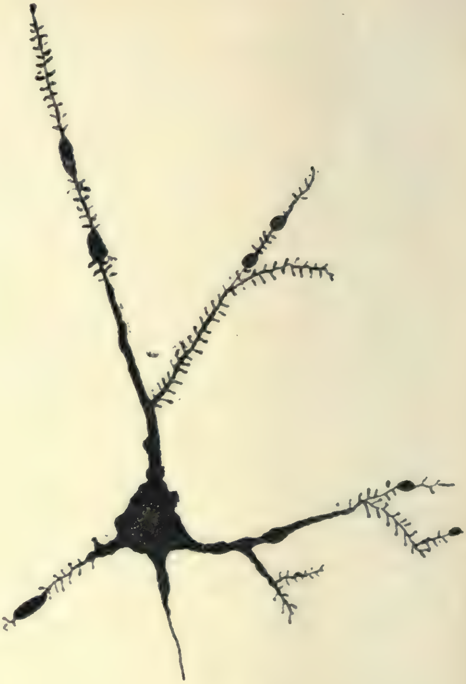

FIG. 3.

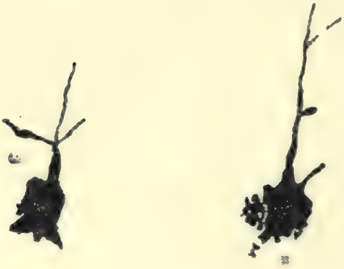

FIG. 2.

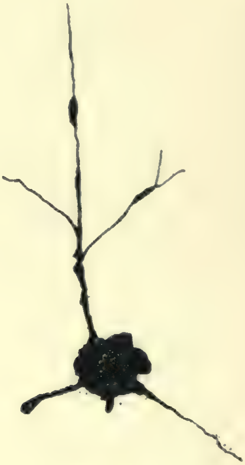

FIG. 4.

Extremely degenerated pyramidal cells from the human brain. Excavation, wasting, and disintegration of the bodies is seen. Also budless swellings on the protoplasmic extensions and thinning and disappearance of the distal parts of the latter.

sharply defined oval, round or rod-shaped masses. They may be shed portions of the degenerated processes which now serve as a contact body for a silver deposit, or they may be simply a precipitate of the latter agent itself, having no structural basis, although the sections as a whole are remarkably free from "blobs."

The primal apical process at, and for some distance above, the cell from which it takes origin is ragged in outline and on one or other side displays a single jagged excrescence or a series of excrescences. Farther up the stem may be in no wise unsound for a short distance; yet sooner or later varicose enlargements are met with bereft of buds and altogether similar in aspect to those already described.

Only rarely in examples of degeneration to the degree portrayed in fig. 3 can an apical dendron be traced to any great distance from its cell. A pointed or globular ending is usual in the upper layers of the external pyramidal lamina. Beyond this, débris marks the alignment of the process. A globular, sharply defined mass, an attenuated black or more lightly stained line or two, or a moniliform thread with scarcely recognisable lateral projections yet remain of the once healthy extension. The secondary branches are in most cases reduced to mere stumps, with here and there along their short course thinned truncated remains of tertiary twigs.

The axon of cells so far involved is yet almost intact. But a swelling of it is to be observed close to the base of the cell from which it takes its departure, quite twice the size of the similar part of the process of the healthy cells. No doubt it portends the absolute loss of the axis cylinder which has occurred in fig. 4 and other cells about to be described.

A still more extreme stage of degeneration is that pictured in fig. 2. The cell body may be roughened in outline and shrunken to one-half or one-third its normal dimensions. It is surmounted by a withered ragged-edged process whose secondary branches have disappeared *in toto*. Such an apical process is of short length and gradually dwindles from its base, and finally ends in an attenuated termination, beyond which lie a few black or deep brown dots and dashes not unlike a "Morse Code;" the probable remains of the once sound structure. Basal prolongations have disappeared almost entirely. Some evidence of the axis cylinder is yet visible, however, in the swollen stump coming off from what was in health the base of a cell; but beyond this the structure is not observable.

The possibility of *post-mortem* alteration is best considered at this stage.

EFFECT OF POST-MORTEM CHANGES.

Pieces of human cerebrum were exposed to conditions as nearly as possible of the same nature as the diseased brain, and then used as control.

Such control tissue showed with aniline dyes a slight over-staining of the achromatin of both the body and nucleus of the cells, but no loss in number of stichochromic particles in the former, or the larger molecules from the latter. The dust grains of the nuclei were also obscured or lost to view in the unusual colouration of the nuclear matrix. The nucleoli, however, were well stained, smooth, round, and sharply defined, and presented none of the aberrant forms and appearances described. No angular particles were visible in the nuclear vesicle, nor was the vesicle itself dislocated except rarely, and its outline was sharp and circular, or oval. Occasionally a cell appeared to be enlarged but never ragged in outline, or with its margin undergoing a process of disintegration. Shrinking or plication was not visible in any of the cells.

When stained by the silver phospho-molybdate method axons were found to retain their position, but were occasionally enlarged close to the cell if the cell itself seemed enlarged. Such enlargement was also noted to be a feature of a few basal and apical protoplasmic extensions. But the withering of cell bodies such as seen in fig. 2, or the dwindled extensions of figs. 2, 3, and 4, were not observable. Nor in any instance were there present the varicosities shown in figs. 1 and 3, except (rarely) at the forkings of the secondary and tertiary branches. Lateral gemmulæ were present and well stained in most instances, but whole stretches of many processes both apical and basal were stripped of them; yet they were to be seen scattered through the section, and may therefore be looked upon as accidentally disengaged from the parts to which they belong. This observation is further strengthened by the fact that, in sections purposely cut thick so that processes might be seen between the upper and lower planes without actually appearing on the surface of the section, the lateral gemmulæ were found to be in position, although on account of the thickness of the sections they did not show with the same sharp definition of the thinner sections.

Of considerable importance also is the fact that in every instance observed by both aniline and silver stains the nodal points of support cells and their extensions are healthy. None of the aberrant shrunken forms mentioned in connection with the morbid brain are to be observed.

It seems likely, therefore, that the more marked deformities presented by the cerebral cortical cells in this case are due primarily to the deleterious action of the extreme doses of the bromide. The altered histo-chemistry of the nuclear fluid whereby the dust grains are lost to view may be the result of the *post-mortem* exposure which the encephelon underwent; and so also the occasional enlargement of certain cells and the beginnings of their processes. But the disintegration of the cell margin, the loss of figure, and change in staining quality presented by the nucleoli, the appearance of the foreign angular particles within the nuclear sack, must be regarded as degenerative.

The bulbous enlargements of various sizes, the disappearance from them of the rectangular buds, the shrinking of processes in cells more deeply implicated, and the ragged edges and excavations of cells, together with the withering and distortion of the latter and the disappearance of their axons, can only be looked upon as the work of a violent poison which operated during life. This conclusion is supported by the action of the same poison on the cortical cells of the rabbits as I will now proceed to show.

EXPERIMENTS AND OBSERVATIONS UPON RABBITS.

Four rabbits were used in these experiments which were treated in the following manner:—Rabbit No. 1 received by means of subcutaneous injection 36 grams of potassium bromide in twenty-two days; rabbit No. 2, 40 grams in twenty-four days; rabbits Nos. 3 and 4, 36 grams in twenty-two days.[1] Following upon the first injections the animals became drowsy and inactive, refused all food, and remained in the corners of their cages for a few hours. When disturbed their movements were slow, automatic, and were quickly followed by quiescence and renewed somnolency. At first they recovered full power of mentality and movement, and were lively in action and appetite, in the intervals between the subsidence of the effects of the drug and its next administration. But after the eighth or ninth day, the drowsiness and depression of motility became gradually prolonged, and the animals passed into a state of stupor and began to lose flesh. Fifteen days saw greatly increased depression of both mental and motor functions, and the sensory faculty became so benumbed that the animals no longer protested against the prick of the syringe. The effects of the injection here began to blend one with the other, and the phases of recovery observed at first were not again noticed. From now on the animals scarcely moved unless disturbed, and action following stimulation was purely automatic. The fore legs soon became paralysed, but the hind limbs still retained some power. Reflexes were depressed excepting the conjunctival. There appeared to be no great desire for, nor was there any movement towards, food; yet this would be taken slowly but without relish if placed within easy reach. About the twentieth day stupor of high degree supervened, and two days later it was followed by deep coma, accompanied by complete neglect of food, loss of sphincter control, and absence of all mental and motor manifestations. All four animals presented a picture of total inertia, bar an infrequent and weak prolonged respiration, or an occasional twitching of the nose and ocular muscles.

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[1] At first I experienced considerable difficulty in administering the bromide of potassium to the rabbits on account of its extremely irritant action at the point of injection into the subcutaneous tissues. As I proposed to get the psycho-motor cells affected as early as possible with poisonous quantities of the salt, it was necessary in order to accomplish this, that large daily doses be given. When the extremely irritant action of a concentrated solution of potassii bromidum is called to mind, it may be readily imagined that the animals proved rebellious to 1 or 2 grams injected under the skin at one point, and that inflammation in the neighbouring tissues speedily supervened. Such indeed was the case, but this was escaped from by diluting my solution from the 1—3 strength of the first injections, to 1—9, and then injecting 12 cc. at three different places at the same time once a day. With this change of plan, irritation and inflammation at the point of injection were reduced to a minimum, and after the first ten days no pain seemed to be experienced by the animals, either as a result of the puncture, or of displacement of the tissues which occurred to make room for the fluid.

As now death seemed inevitable if the drug was pushed any further, a halt was made and a chance given to the animals to recover. But during forty-eight hours, the inertia continued, and it seemed that a necessary quantity of the bromide salt had been given to produce a maximum and fatal effect.

To insure a perfect preservation of the finest and most delicate constituents of the cerebral cortical cells and their appendages, the animals were bled to death, and their cerebra placed in either alcohol or Müller's fluid. But a few minutes elapsed between the bleeding and the immersion of the organs in the fixing media.

Chloroform, ether, and other drugs, were purposely avoided in order to evade any possible complication.

As brains Nos. 1 and 2 were intended for examination by the silver method they were placed in Müller's fluid. The control for these was carried through the same fixing solution.

Brains Nos. 3 and 4 were fixed by graded alcohol. The control for these was treated in the same courses of alcohol. At the end of two weeks the organs were passed through celloidin, cut, and stained by Nissl's polychrome and hæmatoxylin-eosin methods.

CHANGES IN THE RABBIT'S BRAIN AS SHEWN BY NISSL'S AND THE POLYCHROME METHODS.

The control brains in none of over three hundred sections show departure from the normal, but this is not so in the cells of the poisoned rabbits.

Some stage of degeneration is so universal that I gave up trying to arrive at the proportion of diseased cells to those yet in health, and therefore reversed the calculation to an estimate of the ratio of sound bodies to those showing atrophy. About three times in every field, as shown by Reichert 1-18 objective, a cell approximating to those of the control cortex could be observed. The average number of cells that appear clearly in any single plane of a field is about fifteen. So that the proportion of normal cells to those in some stages of atrophy is about one to five. This is a rough calculation only, but it will indicate in some measure how widespread has been the action of the bromide, and also that probably no single type of proper cortical cells escaped the effect of the drug.

Cell bodies, their processes, in fact all the constituents of the cells may be involved together, or only one or other.

But a minute description of the atrophy is called for, because of its likeness in many features to that in the human cortical cells, and also for the reason that it places beyond cavil the observations made upon the human brain by Berkley.

Cells in an incipient stage of degeneration are not so numerous as in the cortex of the human cerebrum. But though proportionately not so numerous they are yet present, and indicate what is probably in the cell body, at any rate, the beginning of the poisonous action of the drug. Such cell bodies may be quite regular in outline but more often a slight crenation is noticeable on one or other side of the cell, or the entire margin may be markedly dentate. In the latter case there is a slight colouration of the ordinarily clear hyaloplasm, and therefore an obscuration of the stichochromic granules. But I cannot say definitely that any of the latter have disappeared; compared with those in an absolutely healthy cell in the same section or in the control tissue, they do not seem diminished in number.

The nuclei of such cells have not escaped. Whenever the cell body is affected, no matter in how slight a degree, nuclei show some departure from health. Their membranes are delicately wrinkled in some one sector, and perhaps plicated but well defined in the remaining portions; or the whole membrane may be crinkled, and at the same time deeply plicated at some one point, and irregularly stained.

The internal constituents of the nuclear vesicle have undergone a change, but of slight degree, in these slightly affected cells. The achromatin is blurred and has lost its high degree of refractility. Fine nuclear dust is obscured by the over-colouration of its matrix, and the larger molecules are reduced in number. Only rarely can more than two or three particles be observed in the sack. Nucleoli are deeply stained throughout but are no longer round and clearly defined. Their edges are now delicately granular either in one or more short segments or throughout.

Cell bodies more altered than those described above are very common. In fact they form the great majority of all unsound cortical cells. The outer edge of a cell may be markedly wrinkled throughout and in some cases show a deep plication which has bent the nuclear sack inwards at the point of impingement. The cell margin also is deeply stained, but the remainder of the hyaloplasm may in such a case be clear or only delicately tinted. Not always, however, is the over-staining of the edge of the cell uniform throughout; its periphery may show in parts a finely granular refractile substance which has escaped the persistent action of the reagent and which probably foreshadows the ultimate breaking down of the whole cell. The hyaloplasm not yet absorbed is clear but almost devoid of chromatin granules.

The nuclear ring is crenated or deeply plicated. In many instances it is not discernible in its entire circumference. The beaded appearance of slightly involved cells no longer prevails. Now, comparatively large portions of the membrane are finely granular and etiolated and have interposed between them deeply stained smaller portions, or badly defined globules. In some instances the greater part of the nuclear margin is scarcely distinguishable but seems to be disintegrated and about to coalesce with its matrix and the yet remaining hyaloplasm.

The nuclear fluid is altered in quality; it is not highly refractile, it does not stain deeply, but faintly, and finer dust particles have either absolutely, or in great measure disappeared, but there is on the other hand an increase in the size and number of the larger molecules in many nuclei. Eight or nine is not an unusual number and they range in size from twice to three times the size of normal nuclear molecules as seen in control specimens. In shape they are never round, but angular. They are similar to the large foreign particles recorded in connection with the altered nuclei of the human cerebral cells.

The nucleoli may be oval, eccentrically situated and intensely stained at one or both ends, the intervening part being pale and finely granular. On the other hand they may be faintly stained and swollen to twice or thrice their normal dimensions, and with delicately granular margins of the same degree of colouration as their bodies. The latter feature seemed to be due not to an aggregation of nuclear dust, but rather to a complete granular transformation of the nucleolus itself. In such a case a nucleolus, could it be seen in its entirety, would present a punctate surface, but in cross section it presents only a delicately granular margin. Prevalent, but less common than the above described state, is a nucleolus stained so that it resembles a crescent. One aspect may be coloured as a sharply defined crescent, but never so deeply as a healthy nucleolus. The remainder of the structure is in such a case scarcely obvious because it is pale and ill-defined.

This state of the nucleoli appears to be the precursor of the foreign angular particles that I find both in the human and rabbit cells, and which were first described by Berkley. First of all there seems to be a swelling and spongy change of the nucleolus, hand in hand with this an alteration in the chemical property of the structure whereby it stains aberrantly, and finally a disintegration into several particles which now separate and lie in the nuclear matrix as the foreign molecules.

When describing the state of the nucleoli in the human cortical cells it was pointed out that occasionally they were enlarged and arrayed in short club-shaped or pointed spurs.

On the whole such a state was rare. But in the cells of the lower animals the condition is met with often.

A still more extreme stage of degeneration of the whole of the cell body is not infrequent. In about every twenty, or twenty-five cells one is to be seen in a degree of degeneration not far removed from total destruction. All components are embraced in the wasting. A thin deeply stained, or irregularly stained, plicated membrane is all that is left of the body plasm and this encompasses an intensely dyed and puckered nuclear sack. The membrane appears to be formed of altered hyaloplasm. In many cases it is granular and pale in parts, or may have broken down into a loose *débris* which now spreads outward into the pericellular space. The crenated margin of this membrane may stretch across the angles of a plicated nucleus, thus forming tiny bays in which rests a minute quantity of clear hyaloplasm. Or the latter may not be wholly amorphous, but contain a granule or two similar to, but larger than, the stichochromic particles of the sound cell.

In yet other instances the altered cell plasm, very deeply stained, may closely embrace the shrunken deeply stained nuclear vesicle and be almost indistinguishable from the latter. The form of the whole cell may be long and elliptoid, oval or round. No dust grains are visible in the nuclear fluid of such cells but the whole achromatin is intensely stained and may contain the large angular particles that obtain in cells already described. When they are found they are of even deeper hue than the matrix in which they lie.

It will be noticed that there has been no mention here of swollen or enlarged cells similar to those found in the human cerebral cortex. I looked carefully for them but saw none. The atrophy of cells in the rabbits' brains has been widespread and extreme, but it appears from its inception to its most extreme degree to be one of shrinking, and chemical and molecular rearrangement. It is doubtful, therefore, whether the enlargement of the numerous cells in the human case is an actual phase of degeneration induced by the action of over-doses of the bromide salt.

In many ways, however, the atrophy observed in the lower animals agrees with that seen in the human cortical cells. The altered reaction to dyes of both body and nuclear plasm, the disappearance of stichochromic granules from the former, and of dust grains and larger molecules from the latter, the appearance of the foreign angular particles in the nuclear vesicles in both, and the aberrant staining and form of the nucleoli of each are changes of a like kind, and point to an irritation of the cells by a similar agent. So also do the more extensive alterations in the cells :—the marked shrinking of both body and nuclear sack, their ragged edges, and their partial breaking down and absorption.

True, there are divergencies in the type of degeneration in the two species of cells possibly explained by the differences existing between the human and animal cells. In the rabbit eccentricity of nuclei is scarcely ever recognised, while such prevails widely in the human cortical cell.

Here as well as in the human cortex many nodal points of the support tissue are unsound. Scattered indiscriminately through the cortex are many cells with markedly wrinkled outlines and amorphous deeply stained bodies. These present no constant form, but may be deeply notched on one or two opposite sides or irregularly kidney-shaped. On the other hand, a smaller number are large, much larger than any in the control sections, pale, and their granules scarcely stained. It is a noticeable fact that here as in the human case, below the innermost cortical layer few affected support nuclei are to be seen.

Blood-Vascular Changes in the Rabbit Cerebra as shown by the Anilines.

Contrary to the conditions in the human pia-mater I found on exposure of the rabbit cerebra that there was no injection or œdema. The brain surface was anæmic and dry. It is quite possible that the mode of death of the animals accounts for these conditions; for they were bled to death.

Microscopic examination of the vessel walls shows the swollen endothelium, and bulging nuclei, which in some instances project and come in contact with the opposite wall. But the capillaries appear collapsed and are almost devoid of corpuscular elements. The median sized and larger vessels are also nearly empty but their endothelial nuclei are swollen, bulging, and deeply stained. Their size and affinity for dye is far in excess of those of normal endothelium. The peri-vascular spaces are in most instances full of a fine granular detritus. But on the whole the disturbance in the blood vascular system of the pia and cortex is slight and not so noticeable as in the human brain.

HISTOLOGICAL CHANGES IN THE RABBIT CEREBRA AS SHOWN BY BERKLEY'S SILVER PHOSPHO-MOLYBDATE STAIN.

As before, the pyramidal cell will be the chief subject of study. And I may say at the outset to avoid repetition that this cell has to all appearances borne the brunt of the action of the potassii bromidum. It is more often and more extremely affected than those of other form and position, but in those others nevertheless the kind of atrophy is exactly similar to that in the principal psychical cell.

By comparing camera lucida sketches Nos. 5, 6, 7 and 8, with those from the human cortex no great or essential discrepancy may be detected between them. In fact, so closely do they resemble each other that they are almost interchangeable and one might be used to illustrate the other.

Cells not deeply affected by the poison are visible in greater number than in the human case; but this may be apparent rather than real. For in no section of the latter are there so many cells shown as in the case of the animals. Yet the lesion is virtually the same. We find a slightened roughened body such as in fig. 5, or one that wholly normal shows on its processes bulbous swellings altogether similar

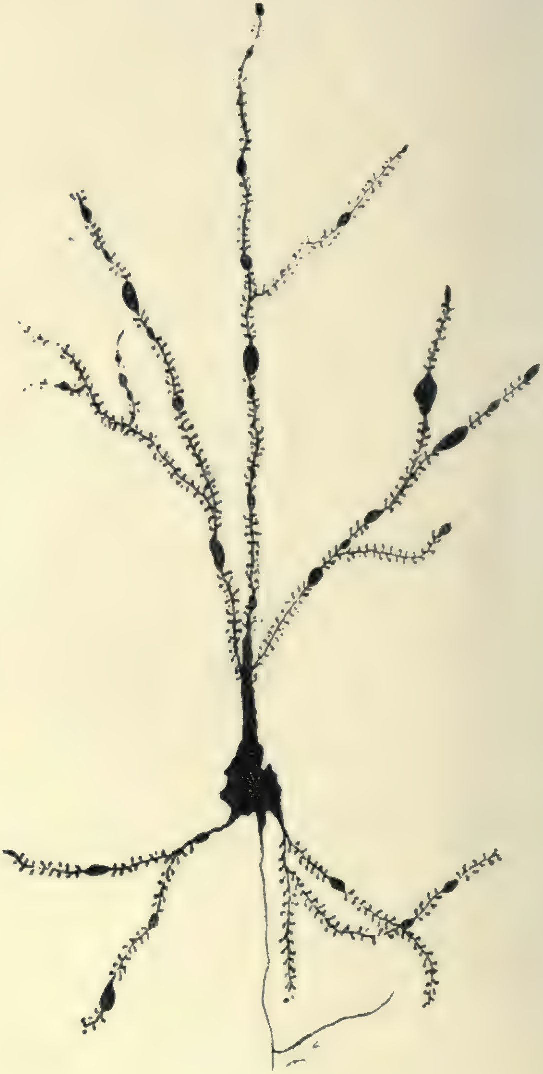

FIG. 5.

Early degeneration of a pyramidal cell and its dendrons in the cortex of the rabbit.

to those observed in the case of the man. In every instance observed the swellings are devoid of buds, and, therefore, contrast remarkably with the apparently normal rest of the dendrons. Here the artificial disengagement of the lateral projections which has occurred widely in the human cortical cells has not taken place, for by more careful management the tissue was not allowed to over-harden.

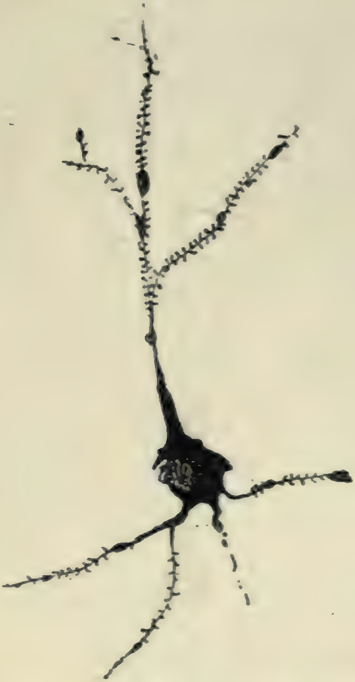

FIG. 6.

Pyramidal cell of the rabbit, showing excavation of the body, and wasting of secondary and tertiary branches of protoplasmic extensions. In this, the extremity of the ascending process has wasted.

Fig. 5 shows truthfully the condition of many of the pyramidal cells. As in the human case the primary ascending process and its first and second twigs are more noticeably implicated than are the basal and their branches. Many cells are visible (fig. 6) in which the apical process has dwindled, and the off-shoots are altogether wasted or scarcely recognisable on account of their great attenuation. But either where the processes still exist, or where they have disappeared, we find the characteristic varicosities marking their alignment. These are in many cases plentiful in number and various in size and form.

Along the emaciated protoplasmic filaments (fig. 7), there appears to be a scarcity of, or wasting of lateral gemmulæ; and there are but a few detached ones to be observed in the adjacent tissue. Where sections were purposely cut thick, and with the use of strong illumination, it was determined that even in protoplasmic processes in the depth of the section, the rectangular buds were decidedly small in number, and it was not unusual to see an entire absence of three or four from one side of the stem, while the other side was apparently healthy. They could not have been disengaged by the microtome knife. It is possible of course that owing to some delicate chemical change they have not stained. Yet the cell bodies and the extensions themselves, even when attenuated were deeply blackened, and in perfectly normal cells in any of the sections no lateral buds were detached. It seems possible therefore that the absence of the lateral buds is one phase, and probably an early one in the process that has caused the bulbous swelling, the wasting and absorption of the dendrons and the shrinking or distortion of the cell bodies.

Such a conclusion is borne out by the appearance of cells as portrayed in figs. 7 and 8. For in these the attenuation of protoplasmic prolongations is associated with a thinning out, and finally, in some instances, complete atrophy of the lateral buds. It is needless to point out that in the case of the swellings on the dendrons no matter of what size there is a complete absence of gemmulæ. And it need scarcely again be argued that this phase is degenerative rather than artificial.

Figs. 7 and 8 explain themselves. In them are seen shrinking and atrophy of the cell bodies. Excavation and roughening of the margins is often observable. The primal apical and basal processes are withered and shortened, and

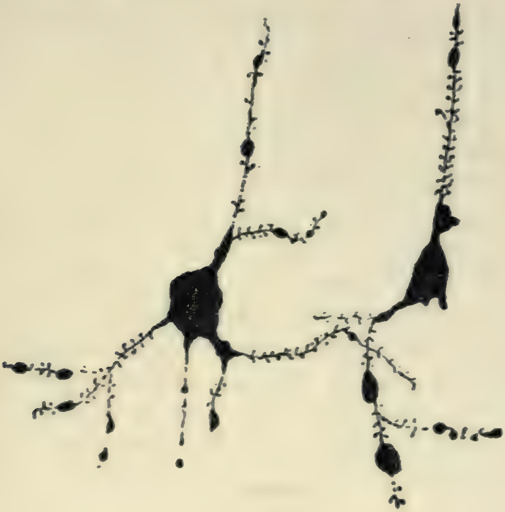

FIG. 7.

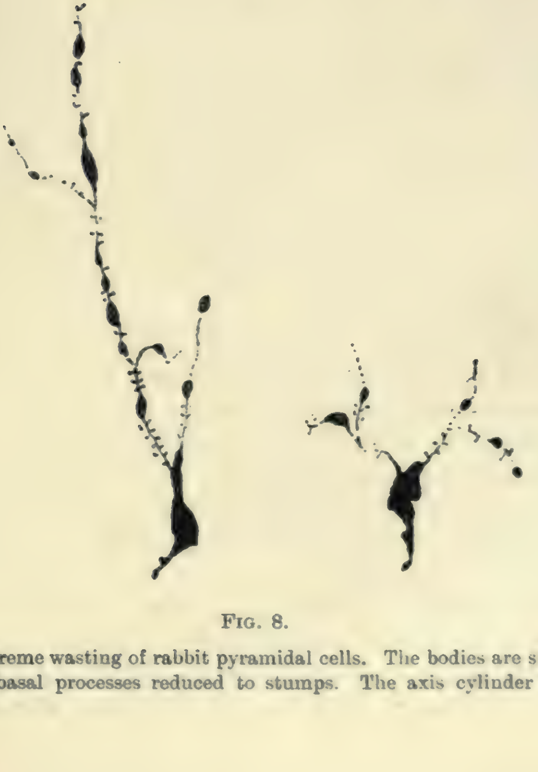

FIG. 8.

More extreme wasting of rabbit pyramidal cells. The bodies are shrunken, and many basal processes reduced to stumps. The axis cylinder has also wasted.

bear upon them bulbous swellings of various sizes. Portions of the distal part of the extensions are visible in many instances as dots and dashes.

Still more extreme degeneration is not uncommon. All the processes of the cell may have perished from the poisonous action of the potassii bromidum. In such a case the cell body is reduced in size, distorted, and roughened in its margin, is not far from final disintegration and disappearance. How many have actually been absorbed it would be as unwise to speculate upon here as in the human case. But it may be concluded that some have absolutely disappeared considering the low ebb to which many have been brought. This may have taken place, and yet its influence on the economy of the cortex be of little importance. For it is not to be believed that the absolute removal of a small number of cells would be productive of so great a psycho-motor disturbance as the slight affection and therefore disturbance of function of a great many.

The axis cylinder in the experimental cases is worthy of consideration, and permits of more complete observation than in the somewhat over-hardened human cortex, because of its not having been accidentally detached. In cells such as pictured in fig. 5, the axis cylinder processes are sound from their point of departure from the cell body to where they become lost to view in the medullary centre of the hemisphere. Their collaterals are likewise normal so far as they can be traced.

But in instances of degeneration such as portrayed in fig. 6, and in all types of more advanced atrophy, the axis cylinder shows degeneration either early or advanced. Seldom are swellings to be seen similar to those upon the dendrons. In all cases where they occur they are small, rarely exceeding twice the diameter of the structure they affect. Close to the cell body, the calibre is increased far in excess of the normal, and occasionally a corkscrew distortion occurs; but it appears to be a late rather than early phase of atrophy. It is never recognised unless the distal portion of the axis cylinder is deeply implicated or has absolutely disappeared.

The distal portion of the axis cylinder is always most markedly attenuated, and is the first to show signs of solution of continuity. It would thus appear that degeneration of the axial prolongation begins with attenuation more extensive in the distal visible portion and its collateral than in the proximal part of its course; that this progresses to a solution of the distal segments, finally creeping upwards. This seems to be so in such cells as in fig. 6, in which there is the first positive involvement of the neuroaxon.

But in cells the bodies of which are intact as far as may be judged by the silver method the axial prolongation is sound in all its traceable length. But if distal degeneration occurs first, as seems to occur from observations upon atrophic axis cylinders, there is no gainsaying that even in the case of cell bodies which are apparently sound, and yet whose dendrons are withered and varicose, the ultimate ramifications of the axons may be in an incipient stage of atrophy.

No instance of cell degeneration has presented itself in which the body of the cell alone has suffered. If this is in the least affected its dendrons are also involved. The latter may be thin in all their length, the seat of budless swellings, and show signs of a break in continuity, and absorption of their distal observable extremities. On the other hand, I have not failed to see many apparently normal cell bodies whose dendrites are nevertheless the seat of a few small bulbous swellings and slight attenuation. All through the cortex it may be seen that the farthest removed portion of a protoplasmic extension is the first to betray the action of the bromide salt and to become absorbed. Swellings may obtain in any part of its length, but atrophic solution of continuity never occurs close to the cell body without there having been first of all a thinning and absorption of a great stretch of the terminal end of the process. Death is from without inwards in every case. But it is hardly probable that the cell bodies remain unaffected for any great length of time, for marked implication of the protoplasmic extensions, and especially of the primary ascending one and its branches, is associated with a rough edged, shrunken or excavated cell body. Great atrophic involvement of cell bodies is associated with attenuation and absorption of the neuroaxon. It is difficult to decide, however, whether the affection of the ascending process is really primary, or only primarily obvious. There is to be kept in mind the possibility that a slight, unrecognisable implication of the cell bodies may have first occurred and thus rendered all the ramifying processes defenceless against the poisonous action of the salt.

Another point worthy of note is that there appears to be no relation whatever between the degree of atrophy in either proper or support tissue and the position or size of the blood vessels. Those cells which lie close to a blood vessel of any size are not more involved than others relatively far distant. The salt evidently finds every part of the cortex equally accessible, although its greatest degenerative effect is beyond doubt upon terminations of all nerve cells and more especially those of the chief pyramidal layer.

Conclusion.

There has occurred then beyond doubt a change in the proper cortical cells and their appendages, and to a slighter degree in the support and vascular tissue in both the man and rabbits as a result of the action of pronouncedly toxic doses of potassii bromidum. In the case of the man there is reasonable room for doubt as to whether the whole of the abnormal changes presented by the nerve cells is entirely due to the influence of the salt. But after making all possible allowances for fortuitous complications, there yet remains a series of changes in them which cannot be attributed to a cause other than the suspected one; and this is substantiated by control observations, and by experiments upon the action of the same cause on the cortical cells of rabbits.

If under the same environment and dosage of bromide for the same length of time, there had been a radical or even noticeable difference in the kind of atrophy in the nerve cells and their appendages of any one of the cerebra, it would have opposed such a conclusion and rendered the experiment of little value as a control for the human cortex. Moreover, the severity and extent of the cortical change in both the man and animals is no more or less than what was to be expected in view of the psycho-motor phenomena which they manifested during the period they were under the active influence of the drug. And this leads to the question of the mode of action of potassii bromidum when administered as a narcotic.

It is not disputed that bromide of potash when given in single excessive doses modifies in a marked manner the function of the central nervous system; that on the chief portion of this, the cortex cerebri, it is certainly depressant, interfering with the elaboration of thought processes and lowering the activity of the motor cells.

The *ante-mortem* phenomena, watched in the case of the man under conditions where there could be no possible bias, and in the case of the rabbits with a full knowledge of the force at work, corroborate fully the above opinion. The microscopical appearances both in the accidental and experimental cases of poisoning place beyond doubt the fact that toxic doses of the salt repeated at short intervals are capable of actually destroying the proper tissue of the important surface grey matter, upon which in physiological doses it operates not more than as an inhibitant.

Such being the case, the important question arises as to the manner of its action in the cerebral cortical area, whether it affects the bodies of the cells first, and thus the whole of each neurone, or whether it affects the bodies after a primary action on their delicate protoplasmic extensions.

It has been stated that in the cases of toxic degeneration under discussion many cells are observable in both classes of cerebra with normal bodies, but showing signs of atrophy (*i.e.*, bulbous swelling and attenuation), in the distal extremities of both basal and apical stalks and their branches; that on the other hand never can an even mildly affected cell body be seen bearing absolutely sound dendrons; and that in all examples where the latter are affected the atrophy in them is proportionately greater as they are followed to their ultimate visible ramifications.

Obviously, therefore, the visible degeneration began in that part of the cell spread farthest removed from the cell body.

Taking it for granted that that part of the cell structure which shows greatest disturbance from the action of any hurtful agent is the part upon which such an agent chiefly acts, it seems likely that potassii bromidum as a poison, operates principally upon the protoplasmic processes of the cells rather than upon the bodies themselves. I regret that I did not graduate the dosage of the salt in the experimental cases, for by so doing I might have determined the incipient point of the atrophy; this must be left for future observation.

But it seems justifiable to conclude that the drug in its mild action operates upon the ultimate termination of the protoplasmic extensions, and that therein lies its power of inhibiting intellectual and motor processes; first, by creating an undetermined chemical change in the delicate unprotected terminal knobs of the protoplasmic extensions and of their gemmulæ, which renders them obtuse to impressions brought to them by fibres from the periphery and from other parts of the encephalon. This does not necessarily imply an alteration in the degree of contiguity of the amyelinic knobs of either incoming or receiving cell extensions, but only a "psychical numbness," as it were, analogous to the physical numbness which occurs in the end-organs in the surface of the body under the effects of special agents. A widening of the normal breach between the terminations of the dendrons and gemmulæ is probably a later manifestation of the action of the drug, and may be due to its secondarily affecting the cell body, thus causing a general retraction of all ramifications of each individual neurone which Cajal claims is one of their attributes. The continued action of the drug on the dendrons in such a state would soon lead to the changes described in these specimens.

BIBLIOGRAPHY.

(1) *Neurol. Centralblatt*, 1887.
(2) *Arch. f. Psych.*, Bd. 24, 1893.
(3) "Sulle fine alterazione della corteccia cerebrale in alcune malattie mentale," *Roma*, 1894.

(4) *Berlin Klin. Woch.*, No. 14, 1894.
(5) *Arch. de Neurologie*, 1894.
(6) *Ibid.*
(7) BRAIN, 1894.
(8) *Bollet. della Soc. Med.-chir. di Pavia*, 1895.
(9) *Johns Hopkins Hospital Reports*, vol. vi., 1897.

NOTE.—To Prof. J. Geo. Adami I am indebted for many useful hints in this work.

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THE MORBID ANATOMY OF A CASE OF LEAD PARALYSIS. CONDITION OF THE NERVES, MUSCLES, MUSCLE SPINDLES AND SPINAL CORD.

BY E. E. LASLETT, M.B., B.SC.
House Physician to the Royal Infirmary, Liverpool,
AND
W. B. WARRINGTON, M.D., M.R.C.P.
Demonstrator in Pathology, University College, Liverpool, and Assistant Physician to the Hospital for Chest Diseases.

WE are indebted to Dr. Caton for permission to record the following case which presents some features of interest especially in the condition of the spinal cord.

CLINICAL HISTORY.

F. K., aged 28, house painter. Admitted to the Liverpool Royal Infirmary, January 14, 1898. He had been engaged in his occupation since his twelfth year and two years ago suffered from colic. Eighteen months ago he noticed weakness and wasting of the hands which attracted his attention before the wrist drop, the prominent feature on admission.

Condition on Admission.—Patient a rather emaciated man with an anæmic, sallow complexion, and puffy eyelids. The body was covered with numerous small purpuric spots. There was a characteristic blue line on the margin of the gums. The heart showed evidence of dilatation and hypertrophy. Urine sp. gr. 1012, with some albumen and granular casts.

NERVOUS SYSTEM.

Upper Extremities.—These showed marked general muscular wasting and dropping of the wrists was present on both sides to an equal extent.

The interossei and intrinsic muscles of the thumb were distinctly atrophied.

Wasting, accompanied with complete loss of power, existed in the extensors of the wrist and fingers and thumb.

There was no marked wasting in the flexor muscles of the wrist and fingers.

In the upper arm the triceps and supinator longus were markedly wasted, the biceps somewhat but to a much less extent.

The deltoid and pectoral muscles were also greatly wasted, while the trapezii and spinati muscles were practically normal.

Lower Extremities.—The vasti interni and peronei muscles were wasted, but no other muscles were especially affected although much general weakness was present.

Electrical Irritability.—The extensor muscles of the wrist and fingers did not respond to either the faradic or constant current. The small muscles of the thumb, however, gave the typical reaction of degeneration with the galvanic current.

Sensation.—Slight impairment noticed in the skin over the hands.

Reflexes.—The knee jerks were obtained with great difficulty.

Examination of the rest of the nervous system showed no abnormality. About one month after admission pulmonary complications set in to which the patient succumbed in a few days.

AUTOPSY (48 *hours after death*).

The lungs were found to be extremely congested and œdematous and the heart markedly hypertrophied and dilated. The kidneys were typically granular and contracted. The brain was pale and œdematous. The spinal cord and certain muscles and nerves were reserved for microscopical examination.

PERIPHERAL NERVES AND SPINAL ROOTS.

(1) *Posterior Interosseous Nerve* (see plate, fig. 1).—This was treated with half per cent. osmic acid, and a piece completely teased. *Five* of the nerve fibres were in this way seen to show breaking up of the myelin. The remainder of the fibres which took the osmic stain appeared normal.

Another piece was embedded in paraffin and cut transversely. The sections showed marked atrophy of the nerve fibres, quite half having disappeared; on the other hand those fibres which took up the stain presented the normal black ring and did not show any indications of an acute change, with the exception of a few which appeared swollen.

(2) *Ulnar Nerve.*—Treated in a similar way. No fibres were seen in process of degeneration, but in cross section considerable atrophy was found, but less marked than in the case of the posterior interosseous nerve.

(3) *Spinal Roots* (see plate, fig. 2).—The roots of the 6th 7th and 8th segments were examined by the methods of Marchi and Schäfer.

The posterior roots appeared quite normal.

The anterior roots of these segments in each case showed distinct atrophy of nerve fibres. This was marked in the 7th root, but in none was the atrophy equal in extent to that found in the posterior interosseous and ulnar nerves.

Muscles.—Pieces of the extensor muscles of the hand, and of the interossei were hardened in Müller's fluid, part stained with borax carmine, and part by Schäfer's method. The change in the *extensor muscles* was extreme. Certainly over 90 per cent. of the fibres were atrophied, the majority were not more than 15 μ in diameter. They retained, however, their cross striation, and with the exception of a few fibres no evidence was found of an acute fatty change. The connective tissue was much increased. The interossei were very much less altered.

Muscle Spindles.—In two previously recorded cases of peripheral neuritis these have been examined. In Batten's[1] case both the intrafusal muscle and its nerve were normal, in Gudden's[2] case they both showed pathological change.

In our case they were quite unaltered, presenting the normal features pointed out by Sherrington. Owing to the atrophy of the surrounding muscles the spindle fibres were now considerably the largest seen. The intrafusal fibres

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[1] Batten, Brain, Parts 57 and 58, p. 171, 1897.

[2] Gudden, *Arch. f. Psychiatrie*, Bd. 28, p. 697, 1896.

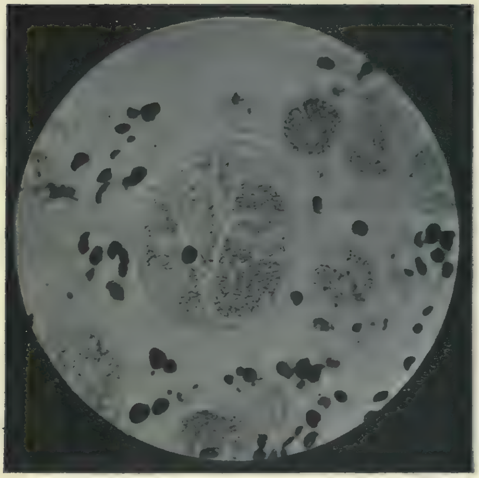

FIG. 1.

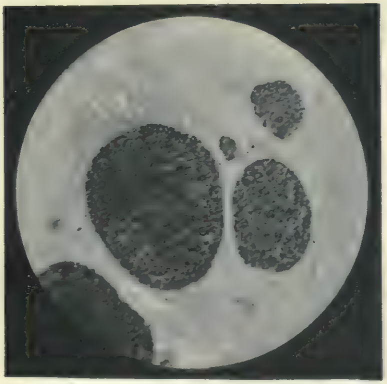

FIG. 2.

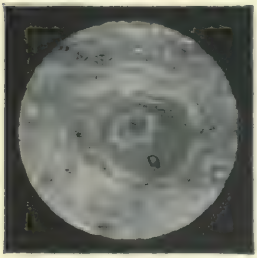

FIG. 3.

retained their striation and at the equatorial zone of the spindle showed the usual numerous central nuclei.

In every case the nerves in the spindles were normal (see plate, fig. 3).

Spinal Cord.—Pieces of the spinal segments from the fourth cervical to first dorsal were examined by the Marchi and Weigert methods of staining and were found to be throughout normal. The fourth to seventh segment inclusive were also stained by the methylene blue and erythrosin method of Held[1] for the purpose of studying the condition of the nerve cells. In the fourth and fifth segments nearly all the cells showed the usual normal structure with a central nucleus and a definite distribution of the chromatic masses giving to the cells generally the well-known striated appearance. A few, however, showed changes similar to those found in the sixth and seventh segments where many cells showed an obvious deviation from the normal condition; in these cells the nucleus was often found placed very eccentrically and also on which fact we lay more stress, the Nissl bodies were dispersed over the body of the cell in the form of fine powder and often in the central mass of a cell an actual disappearance of the blue colouring matter was noticed, the result of a process of chromatolysis. The proportion of altered to normal cells was about one in four, they were found equally on both sides and had no special distribution among the various cell groups (figs. 1 and 2).

Remarks on the above Observations.—Although lead may primarily affect the central nervous system there is sufficient evidence in this case to show that the change began in the peripheral nerves. Firstly, the clinical picture is that of a multiple neuritis, whereas when the disease is primarily central the type is more that of a progressive muscular atrophy.

Secondly, the excess of change in the peripheral nerves over that observed in the anterior roots points in the same direction. The neuritis is probably of a parenchymatous nature as indicated by the condition of the few fibres seen degenerating and by its marked limitation to the efferent nerves, the fibres in the muscle spindle which are derived

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[1] WARRINGTON, *Journal of Physiology*, xxiii., 1898.

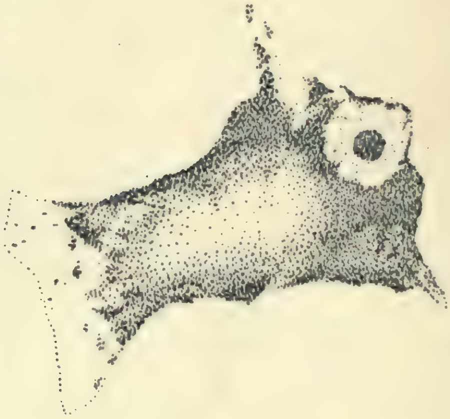

FIG. 1.

An altered anterior cornual cell showing chromatolysis in the centre and eccentric nucleus. The base of the cell is pigmented (×1000).

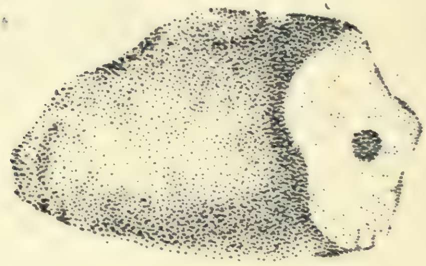

FIG. 2.

Another cell showing a similar condition.

from the posterior roots being throughout normal. As regards the condition of the spinal cord; the introduction of a more delicate method of staining has made it possible to detect more minute structural changes than the older methods were capable of demonstrating. It has now been amply shown that many poisons, and among metals notably those of arsenic, antimony and lead when administered to animals, produce fairly constantly changes in the nerve cells. Nissl[1] and Schäfer[2] have studied the changes produced by the administration of lead and practically agree in their description of the altered cells when stained by the methylene blue method.

The process consists essentially in a disintegration and dispersion of the chromatic bodies and shrinking of the nucleus; in more advanced conditions a chromatolysis sets in so that ultimately a mere palely stained shell-like mass represents the cell.

Such changes probably are due to the direct action of the lead on the central nervous system, but with the existence of multiple neuritis a second factor has to be considered in explaining any alteration of the cells of the spinal cord. After the work of Nissl and others it may be accepted that loss of continuity of the axon is followed at an early period by changes in the corresponding cell. Such changes have been shown by one of us[3] to be especially pronounced after division of an anterior root.

Hence in the above case where the anterior roots of sixth and seventh segments show some markedly atrophied fibres it has to be considered whether the changes in the spinal cord are the result of the direct action of the poison, or if, on the other hand, they are secondary to a lesion of the axis cylinder producing the "réaction à distance" of Marinesco.[4] This observer indeed maintains that the histological features presented by the nerve cell are in general characteristic of a "primary" or "secondary" lesion.[5] A study of the changes in structure occurring in nerve cells in various pathological conditions does not lead us to believe that the histological features alone offer distinctive evidence of their origin. Nevertheless, the condition of the nerve cells in which together with chromatolysis, the nucleus is eccentric, is rarely found except as a consequence of an interference with the afferent or efferent impulses.

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[1] NISSL, *All. Zeitschr. f. Psych.*, Bd. 48, p. 675.

[2] SCHAEFER, *Neurol. Central.*, p. 148 and 900, 1894.

[3] WARRINGTON. *loc. cit.*

[4] MARINESCO, *Revue Neurologique*, March 15, 1896.

[5] *Comptes Rendus de la Société de Biologie*, January 25, 1896.

This, combined with the fact that in our case the altered cells are almost absent from the fourth and fifth segments but found in the sixth and seventh segments the anterior root fibres of which show some atrophy and from which the much atrophied posterior interosseous nerve is derived, point distinctly to the inference that the changes described in the spinal cord are the result not of the direct action of the lead, but as a consequence of the altered state of functional activity following atrophy of the axons of these cells. In cases of polyneuritis from other causes Marinesco[1] and Ballet[2] have recorded similar changes in the nerve cells of the spinal cord which they regard as being due to this cause. These facts, combined with the experimental evidence, indicate that a more extended view must be taken of the pathology of multiple neuritis. The lesion, though primarily peripheral, may be followed by secondary changes in those cells of the spinal cord or of the posterior ganglion (*vide* Lagaro[3]) whose axons have suffered a destruction of continuity.

Note on post-mortem changes.—Our experience with the Nissl stain enables us to believe that when moderate precautions have been taken, the material from the autopsy room gives fairly reliable results. The manner in which the smaller cells stain is often uncertain even in perfectly fresh tissues, but the larger cells of the spinal cord and cranial nuclei seem, with the exception of shrinkage, to retain their normal characteristic staining features for a considerable time after death.

In control examinations of the spinal cord made for the purpose of this paper, no cells comparable to those above described and figured were found.

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[1] *Comptes Rendus de la Société de Biologie*, January 25, 1896.

[2] *Le Progrès Medical*, June 27, 1896.

[3] *Rivista di Patologia nervosa and mentale*, 1896.

Barbacci and Campacci[1] have investigated in a rabbit the condition of the spinal cord cells at varying periods after death, and their descriptions indicate a quite different appearance to anything found by us.

DESCRIPTION OF PLATE.

FIG. 1.—Posterior interosseous nerve (Osmic stain) × 450.

FIG. 2.—An anterior root (Weigert-Pal stain) × 450.

FIG. 3.—Normal muscle spindle surrounded by degenerated muscle (Weigert-Pal and Eosin stain) × 1000.

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[1] BARBACCI and CAMPACCI, *Referat Neurol. Centralb.*, p. 1042, 1897.

NERVOUS SYPHILIS;

With a Critical Digest.

BY W. JULIUS MICKLE, M.D., F.R.C.P.LOND.

THIS article is supplemental to a former one in BRAIN.[1] The present material will be dealt with under the headings of:—

Pathological Anatomy.
Anti-Luetic Remedies.
Pathology.
Para-syphilitic Affections generally.
Para-syphilitic Epilepsy.
The Pathogenesis and Relations of Functional Nervous Diseases in Syphilis.
Multiple Nervous Syphilis.
Hereditary Nervous Syphilis.
Ordinary Spinal Syphilis.
Relations between Syphilis and Tabes.
Insular Sclerosis and Syphilis.

I.—PATHOLOGICAL ANATOMY.

Macroscopically, some of the lesions produced by syphilis have more or less distinctive characteristic features, as, *e.g.*, gumma or gummatous infiltration. Other of the lesions are more or less suggestive of syphilis, especially if taken in relation to the seats of election, the patient's age and medical history, the general excretory, secretory, and blood and tissue conditions of the affected individual. Still other of the lesions in appearance are entirely common to syphilis and other morbid states.

Microscopically, much the same statement holds good, but with less assurance of definite and accurate character, and none of the appearances absolutely pathognomonic. Even gumma is merely one of the "granulation tumours"—a member of a class;

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[1] BRAIN, Spring Number, and Summer Number, 1895.

its cells—chiefly round cells—either close-packed or enmeshed in connective stroma, and undergoing regressive change.

Stress, indeed, has been laid upon "round-cell infiltrations" in or about blood-vessel walls, in meningeal tissues, invading nerve-roots, or cord, or brain, and consisting of cells which possess little protoplasm and single round regular nuclei. But the bare microscopic appearance seems to be insufficient for differential diagnosis of syphilis. The situation and tendencies of the infiltration add a degree of certitude, *e.g.*, the syphilitic infiltration tends very strongly to over-pass the meninges and invade nerve-roots and cord or brain.

Particularly in the pia of brain or cord, also in cord and brain substance, and in cranial and spinal nerve-roots, may occur small foci of round cells. Such accumulations represent potential gummata, and may end as ordinary syphiloma; or in them spindle-cells may come by transformation of the original round-cells, and thus a fibro-cellular structure appears.

Furthermore, syphilitic change of blood-vessel walls may ultimately lead to roughening of the inner lining, with vascular narrowing and tendency to occlusion; these in their turn may lead to thrombosis, and thrombosis to still graver disorder and defect of circulation, or even to ischæmic local softening or necrobiosis, especially in the brain; or the syphilitic blood-vessels may rupture and give vent to hæmorrhage, a condition more apt to affect the spinal cord and its coverings.

A strongly marked feature is the conjunction in some cases—the separate occurrence in others—of syphilitic change of outer, middle and inner coat of blood-vessels supplying meninges or nervous substance.

Thus the alliance of peri-, meso-, and endo-arteritis (or vasculitis), the invasion of nervous substance by meningitic processes, the existence of early gumma (or potential gumma) of meninges or nervous system, and the variety of form, association and succession of symptoms due to changes of nervous substance—all go to clinch the diagnosis of syphilis.

Moreover, an alteration of nerve-cells or fibres—of any part or the whole of individual neurons—may arise, variously, from widely different conditions brought about by syphilis; *e.g.*, the syphilæmia; the circulatory disorder due to vascular and other changes; the local mechanical effects of compression by cell-accumulation or tumour; these and the chemical results of invasion by syphilitic infiltration; the secondary systemic degenerations, whether of Wallerian type, or of the so-called "retrograde" type sometimes observed, and presenting the aspect of a system degeneration.

Thus syphilis affects the nervous system either *directly*, affecting the parenchyma and degenerating the neurons—early or late in its course;—or it affects the nervous elements *indirectly*, affecting them only secondarily and by virtue of the effects of inflammation of vascular, connective, and lymphatic tissue-elements. For example, in the cord, syphilis usually leads to inflammation, &c., of vascular walls, especially of veins and small vessels, to diffuse general infiltration of connective tissue of meninges, to irritation of surfaces laved by cerebro-spinal fluid, and is characterised by miliary gummata of meninges vessels and cord; the nervous parenchyma and neuroglia being affected secondarily, and owing either to (*a*) imperfect nutrition, or (*b*) to mechanical effects of the lesion (Sottas), or (*c*) to both. Yet under the malign influence of syphilis the neurons may directly wither and decay. By some it is maintained that gumma follows irritation of particular regions in the early stage of syphilis, and the residuum of the original infective virus lying dormant in one or another part may be re-awakened into activity by functional hyperæmia, by the effects of injury, or by conditions weakening the protective and immunising forces of the organism.

Tuberculous lesions of central nervous system are often very similar to the gummatous ones of syphilis. But the history of the case, the absence of tubercular lesions elsewhere, and of tubercle-bacilli in the infiltrations or growths affecting meninges and nervous substance, often at least, mark the syphilitic nature of the lesion. When there is a tubercular mass, or when there are tubercles, assistance may be gained from the age of the patient, the site of tubercle, the presence of giant-cells, the marked tendency to caseation.

Sarcomatosis of meninges may be very similar to syphilitic infiltration of the same parts. But the sarcomatous infiltration of lepto-meninges of spinal cord shows extremely little tendency to invade the cord itself or the nerve roots outside the cord; therein it contrasts very strikingly with the great tendency of syphilitic meningeal infiltration to invade or compress cord and extra-medullary nerve-roots.

On the relation between tabes dorsalis, general paralysis and spinal cord syphilis, Nageotte has attempted to bring them all into line, to maintain that in all three are certain chronic inflammatory changes, which, according as they preponderate in this or that particular part of the nervous system, are concerned in the production of tabes, or of general paralysis, or of syphilitic myelitis: there being in all these three a diffuse meningo-myelitis, first attacking vascular and connective tissues, and consisting of a round-cell infiltration; the nervous elements of the cord being affected secondarily. This process chiefly attacks the brain-cortex in general paralysis; the spinal nerve-roots in tabes; and in syphilitic myelitis the diffuse lesion is intense in foci of election. The infiltration is described as embryonic, and as beginning in mesodermal elements of the cord. Yet it does not follow that syphilis is always the effective agent in these diseases. He believes that the simple exaggeration of this diffuse morbid process in special points suffices to create the one or the other nosological species, and that the characteristic vasculo-connective lesions of each of these diseases—transverse neuritis for tabes; meningo-encephalitis for general paralysis; more or less circumscribed meningo-myelitis for the different kinds of spinal syphilis—probably form in relief on a common basis of diffuse inflammation, which is of the same nature in each of them and in some sort serves them as a bond of union. "The lesions of the cerebral cortex, as cause of general paralysis; those of nerve-roots, as cause of tabes; and the localised plaques of syphilitic myelitis, are only the exaggeration of that diffuse lesion in some points" of election.

Distinct as is the fundamental morbid process, and in the (perhaps great) majority of instances connected with syphilis, yet it cannot be exclusively attributed to that agency.

On the question of lesions of nervous system in the secondary and tertiary stages of syphilis, Brasch points out that neither the character of the nervous pathological changes, nor their site, distinguishes the one of these stages from the other; that there is no real, fundamental difference between the lesions of the central nervous system in "secondary" and "tertiary" syphilis.

Before summing up, let us refer for a moment to

THE PATHOLOGICAL ANATOMY OF SOME OF THE SO-CALLED PARA-SYPHILITIC OR META-SYPHILITIC AFFECTIONS OF THE NERVOUS SYSTEM.

Presently, we must speak of para-syphilitic diseases. If we now compare with the genuine and recognised nervous lesions of syphilis those (if any) found in so-called para-syphilitic affections it is seen that the latter are either:—

(*a*) Dynamic dispositions without marked, or at present clearly distinguishable, anatomical lesion of nervous apparatus, or:—

(*b*) Those with appreciable lesions: possessing, therefore, a distinct pathological anatomy.

The first do not come directly in question here, and therefore we pass on to the second, namely, to those of the para-syphilitic nervous affections which possess distinct anatomical lesions. These last are examples of nervous lesions apt to occur in infectious diseases of long slow course, and consist partly of forms of arteritis, phlebitis and sclerosis; usually, or often, more or less scattered in distribution; and partly, and even more strikingly, of various degenerations.

Conjunction or combination of the morbid dynamic and of the morbid anatomical conditions produced by syphilis, probably accounts for the destructive effect of the disease on fœtal or infantile life; and the erection of this effect into a third department of para-syphilitic results is unnecessary.

Then again, some importance is conferred on the so-called "retrograde" secondary degenerations of nervous system (*e.g.*, of posterior columns of cord after cerebral lesions) in relation to the fact that cases taken to be examples of "primary" degeneration are sometimes in reality of the secondary retrograde form; and in relation to some so-called para-syphilitic diseases, such as tabes.

Also, in general paralysis, if viewed as a para-syphilitic affection, in most cases we find no particular admitted well-recognised specific luetic pathological change. Nor are syphilitic symptom-groups, clearly established as such, always to be found in general paralysis. Nor are the effects of classical "anti-syphilitic" treatment of very striking value in this disease.

As a rule, too, visceral syphilitic lesions and tabes do not co-exist.

We may summarise by briefly stating that it is necessary to widen the conception formerly held of lesions due to syphilis, so as to include many of non-specific kind, or not formerly considered to be specific. Here may rightly be placed:—

Meningeal inflammation of several kinds, and other than the frankly gummatous form; in fact, a series occupying a wide range between the gummy and the simply sclerosing form of meningitis, spinal or cerebral:—

Various forms of encephalitis and myelitis (non-gummatous) :—

Softenings in central nervous system, of various kinds: whether of vascular, of syphilomatous, or of inflammatory origin :—

Primary degeneration, or destruction, of nerve-fibres in brain, cord, or nerves :—

Primary atrophy of nerves, nerve-roots, or central nerve-nuclei.

II.—ANTI-LUETIC REMEDIES.

Formerly, one outcome of dominant doctrine was to give to mercurials the most important place in the drug treatment of "secondary" manifestations of syphilis, and to rely mainly upon the iodides in the "tertiary" stage of the disease. Syphilis of the nervous system being then considered as by far most frequently a tertiary affection, the iodides took chief place in its treatment. Consequently, in the ordinances of the treatment of cerebral and spinal syphilis, too much was made of iodides, too little of mercury; the latter being possibly the more important of the two.

Most emphatic are the assertions of some on this subject. For example, Neisser declares that mercury is the only known remedy which attacks the virus of syphilis, although whether it does so directly or indirectly is still uncertain; that any other remedies (K.I., *&c.*), are only ancillary, and by virtue of their property of removing cell-infiltrates; and that mercurial treatment properly conducted and suited to the requirements of the case, is quite harmless. He thinks well of Fournier's intermittent method of treatment: prefers inunction and subcutaneous injection to other forms of administration; and treats for four years, giving seven or eight mercurial courses in that space of time.

Relevant to the discussion of this subject, also, is the observation (Brasch) that in syphilis of the nervous system neither the character of the pathological lesions nor their localisation distinguishes the early and late stages of syphilis; syphilitic lesions of the nervous system being essentially the same in the secondary and tertiary stages of the disease.

On the other hand, Stassano asserts that the affection of internal organs in tertiary syphilis results from an additional toxine possessing necrotic powers, thus differing from the toxine of early syphilis. And Kuh lays stress on the usual presentation of superficial and diffuse lesions by *early* syphilis of the nervous system.

A fuller knowledge than we now possess of the pathogeny of "para-syphilitic" affections would shed light on the obscurities of this subject.

With regard to amount of the rival remedies to be used: some advocate free inunctions of mercurials; as, *e.g.*, of *ung. hydrarg.* a drachm daily; and the iodides of sodium or of potassium in doses of 300, 400, 600 or more, grains a day. As a rule the iodides are gradually raised to these large amounts. It has been maintained (Stieglitz) that these large doses are better borne than are the ordinary small doses of a few grains each, and several such in the day. Strained claims of benefit from over doses of mercury have been made; as, *e.g.*, a case by Krauss of brain syphilis treated by large doses of mercury, which were followed by salivation and by mercurial neuritis of all four limbs.

A prevalent view is considerably in harmony with the opinion expressed by Mills that "when the iodides fail, which is very rarely, if success can be attained at all, mercury may be used successfully even for the late manifestations."

Referring to the subject of the powerful syphilitic microbe Stassano points out that in some respects it resembles the bacillus of tubercle, in some the bacillus of diphtheria, and in still others the influenzal microbe. He thinks serums taken from syphilitics are not of any curative value, and advises empirical treatment, as well as certain serums to strengthen the natural defences of the organism effected by phagocytes.

Early Anti-Luetic Treatment, and its Relations to Occurrence, Kind, or Severity, of Nervous Syphilis.

Early cerebro-spinal lues has often an acute commencement and a rapid course; in contrast with the chronic development and tardy process of nervous lesions of the late stages of syphilis. In relation to this is the question of *early* treatment.

Usually, it has been held that early, orthodox, anti-luetic treatment is of great value in the prevention, or mollifying, of syphilis of the nervous system; and that prompt, efficient treatment tells strongly in those directions; that the rapid and unfavourable course in some examples of nervous syphilis is attributable to the insufficiency or absence of early anti-syphilitic (specific) treatment. Closely connected also with this question is that of defective early treatment (primary and secondary stages) as conducing to the occurrence of so-called "parasyphilitic" maladies.

Writer after writer has emphasised the great importance of this *early* treatment in modifying and mitigating the future phenomena of *late* syphilis. Thus, to mention one only, Neisser declares that in his opinion, "the most weighty ætiological factor in the appearance of tertiary syphilides is deficient mercurial treatment in the early stages of the disease."

But, on the contrary, Dr. Joseph Collins endeavours to prove that exudative and degenerative diseases of the nervous system due to syphilis, are not retarded in their appearance by thorough and prolonged administration of anti-syphilitic remedies during the activity of the virus; that this same treatment does not seem to prevent the occurrence of such diseases as tabes and general paralysis; and that cases confessedly syphilitic and receiving only most desultory and incomplete treatment, are not more liable to the earlier development, or to the severe manifestation of these two diseases than are those in which treatment has been thorough. Unfortunately, the statistical summaries given in support of these conclusions are self-contradictory on several points, which therefore remain unsettled by them, even provisionally.

In my previous articles[1] the subject of the *early* occurrence, in cases, of syphilitic affections of the nervous system, was dealt with.

Spinal symptoms and lesions are not infrequent in the secondary stage of syphilis. Brain syphilis may occur before the chancre is healed, as Kuh states; even syphilitic cerebral meningitis. And spinal cord and brain are attacked by syphilis, on the average, the earlier after infection, the later in life the syphilis is acquired.

Worthy of note in relation to this point as to the *time-relation* of syphilis of the nervous system, is the view of Stassano concerning *the severity* of the disease; namely, that this severity is inversely to the adenitis aroused, this last being directly as the strength of the phagocytes and reaction of the organism against the virus; in consequence of which relation syphilis (*cet. par.*) is more severe in adults than in young persons.

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[1] BRAIN, Spring Number, 1895, *passim*. Summer Number, 1895, p. 352.

III.—The Pathology of Nervous Syphilis.

We have already referred to the facts that syphilis can act on the nervous system in two kinds of way (Sottas) (1) *directly*, attacking nervous elements, as in the vague nervous troubles of the secondary stage, and in the late systematic affections, as for example, tabes; (2) *indirectly*, by producing inflammation of vascular, lymphatic and connective tissue elements, to which the alteration of nervous parenchyma is secondary.

In the latter case, for example, in spinal syphilis, and as already mentioned, there are brought about inflammation of vascular walls, especially of veins and small vessels; diffuse general infiltration of the surfaces laved by cerebro-spinal fluid; formation of tiny or incipient gummata of meninges, vessels and cord. From these conditions comes affection of nervous substance and of neuroglia as result either of imperfect nutrition, or of invasion, or of mechanical pressure and other effects. Of these, the first may end in anæmic necrosis; the second, or infiltration, may invade nerve roots or cord substance from the meninges. The degenerative changes produced by the action of circulating toxine, or by the anæmia of nervous substance, may become more important than the vascular lesions. Yet acute cases may be dependent on vascular lesions. The vascular condition often rules the symptoms produced; and in spinal syphilis the veins may be more affected than the arteries.

In nervous syphilis, the primary or early vascular and meningeal changes are attended by general prodromic phenomena; the softening and degeneration which may ensue have the accompaniment of various trophic and paralytic symptoms; and the final sclerotic state usually goes with chronic spastic paralyses.

An explanation offered of the fact that the posterior part of the meninges and cord is usually the one more affected in syphilis, is to the effect that the luetic virus sinks by gravity to the back part of the cord and meninges, in consequence of the predominantly recumbent position of the body.

In an earlier section we have sufficiently discussed the statement that general paralysis, tabes and syphilitic myelitis, all have at their foundation the same chronic inflammatory process.

Bearing on points of interest is the recent contribution of Stassano. He refers to the gradual tendency of parts, especially skin, to become immune to syphilis. And one of his views is that "primary chancre," so-called, is not a local result of the syphilitic virus but a secondary lesion. At the onset, swelling of the inguinal glands forms a first line of defence and a barrier diverting the virus into blood-vessels of the region, and shutting it out from the general lymphatic circulation. The virus takes action in superficial capillaries of the skin and mucous membrane, the formation of chancre resulting.

This view of the secondary nature of chancre is supported by (1) its vascular origin; (2) cases of absent chancre and absent secondary lesions of syphilis; (3) the fact that solution of continuity is not necessary for the formation of a chancre.

Retardation of lymphatic circulation leads to extravasation of lymph and leucocytes, and these last carry the syphilitic microbes and propagate the disease.

Cet. par., severity of the disease is inversely to the adenitis; this last being directly as the phagocytal strength, and that of the general reaction of the organism against the virus.

These views have been called "new." True it is that chancre is often spoken and written of as the "primary" lesion of syphilis, or as marking "primary syphilis." Nevertheless, it is by no means a "new view" to designate chancre a "secondary" lesion, at least not a new view in the sense claimed. The implication that syphilitic chancre is a purely local affection, and that the "secondary" symptoms of syphilis denote the earliest systemic or general empoisoned condition, was contested many years ago, and some of the best teaching was that in (Hunterian) genuine chancre, the slight induration indicated that syphilis had become general (or systemic or constitutional); in a word, it was influentially held that syphilitic chancre was an evidence of "constitutional" syphilis.

Another of his so-called "new" views on the pathology of syphilis is that in tertiary syphilis there is an additional toxine, possessing necrotic powers and causing affections of internal organs; thus differing from the toxine of early syphilis.

The evidence for this is not convincing. The view is not in harmony with the seeming similarity in essential nature of the nervous lesions in the so-called secondary and tertiary stages of syphilis. It is against the conclusions of Neisser. This observer denies that bacterial virulence has been modified in tertiary syphilis, inasmuch as the peculiarity of tertiary syphilides and their slight infectiveness can be explained by the paucity in number of the microbes; and not by their less virulence. And he agrees with Mr. Hutchinson's hypothesis that the tissues in tertiary syphilis are so profoundly altered by the syphilitic virus, that they no longer react to syphilitic bacilli as do healthy tissues, on the same tissue, in the first stage of syphilis.

Two points he keeps separate. One concerns the factors determining the *localisation* of tertiary syphilides; namely (*a*) residual virus, in former site of primary and secondary syphilides, becoming the starting point of tertiary eruptions; and (*b*) traumatic or pathological irritation of tissues, with resulting neoplastic tendency, &c. The other point concerns the general conditions favouring *preservation* of the virus; such, namely, (*a*) as interfere with the normal measure of destructive power on the virus by the healthy organism; or again, (*b*) such as absence, insufficiency, or delay, of anti-luetic treatment.

The question at issue is weighty in relation to so-called "para-syphilitic" affections. One of the hypotheses on the origin of these introduces variety in syphilitic toxines. According to Fournier's statement of it, condensed in the following lines, the specific microbe would produce morbid phenomena, the results of its specific toxine and direct activity; *i.e.*, ordinary syphilis. Then, later on, this same microbe would elaborate a special toxic substance, a toxine, which, infecting the organism in its turn, would produce another succession of very different symptoms from the first, especially in their resistance to specific treatment; "symptoms of which the prototypes would *par excellence*, be tabes dorsalis and general paralysis."

Therefore the present is a convenient place to consider the para-syphilitic affections.

IV.—PARA-SYPHILITIC AFFECTIONS, GENERALLY.

This term Prof. A. Fournier applied to morbid states which, although not of direct specific syphilitic character, nevertheless are in reality products and derivatives of syphilis. According to this, syphilis is not solely and exclusively limited to characteristic luetic lesions and symptoms: for its action on the organism may arouse disorder of the general health, lessen the vital resistance, affect the development of fœtus and infant, and create organic defects or morbid tendencies. These para-syphilitic affections, although not syphilitic in nature, are syphilitic in origin "in the sense that they are the progeny of syphilis, that they are the result of its occurrence, that according to all evidence they would not be produced without it" (Fournier); *i.e.*, in the given examples. Between them and genuine specific luetic manifestations, a well-defined line of demarcation is drawn by two leading para-syphilitic characteristics: namely, that they do not arise solely and exclusively from syphilis, as do the pure genuine specific syphilitic manifestations; and, secondly, that para-syphilitic affections do not show that amenability to anti-luetic treatment, that prompt and lively movement towards recovery, which so often marks the genuine direct syphilitic morbid states.

Following Fournier, his pupil Gastou deals with the para-syphilitic affections, classing them much as follows, although the loose lines are here a little "dressed."

In the first line come tabes dorsalis, pigmentary syphilide; and, less distinctly so, general paralysis, and the amyotrophy of Raymond.

Besides these, are a number of affections which are taken as being very frequently of syphilitic origin. These para-syphilitic affections are:—

In acquired syphilis; of neurasthenic, hysteric, epileptic, or mental type.

Taking these in order, first comes *the neurasthenic group:* acute neurasthenia of the secondary or tertiary stage of syphilis, and its different forms; secondary neurasthenia; hystero-neurasthenia; ordinary neurasthenia (frequent in the tertiary stage of syphilis); neurasthenic headache; neurasthenic pseudo-tabes; neurasthenic encephalopathies (*e.g.*, simulating general paralysis); local neurasthenias, of peripheral, sympathetic or visceral form (dyspeptic, cardiac, genital); monosymptomatic, as local pain; and, finally syphilophobia, perhaps conjoined with morbid fear of the effects of anti-luetic mercurial treatment.

Also classed as para-syphilitic are certain cases of *hysteria*, which may be slight in degree or of medium or of extreme severity, and quite comparable to forms of common hysteria. Other para-syphilitic hysterias are of "partial" form, *e.g.*, paraplegic.

It must be specially noted, however, that Fournier[1] has also described cases of hysteria in the secondary eruptive stage of syphilis as being of purely syphilitic character, easily curable by mercury, and at least in one case cured by iodide.

Para-syphilitic *epilepsy* usually makes onset in late syphilis; is an isolated epilepsy, *i.e.*, not associated with other morbid phenomena of nervous syphilis; is lasting, and does not yield to anti-luetic treatment, although often ameliorated by potassic bromide.

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[1] *Gazette des Hopitaux*, No. 96, 1888, and *Médecine Moderne*, 1891.

Finally, Fournier places here a variety of lypemania as a para-syphilitic mental change.

Forming a second large group, are *the para-syphilitic affections due to hereditary syphilis.*

These have obtained the classification following, which I take to be faulty, and several of its elements wrongly placed.

(*a*) Fœtal cachexy, namely, greater or less inaptitude for life, leading to death *in utero;* or to the birth of wretched miserable infants with little hold on life.

(*b*) Congenital malformations of limbs or fingers, club-foot, spina bifida, cleft palate, hare-lip, abnormal cranial asymmetry, microcephaly.

(*c*) General or partial dystrophies, namely, arrests or retardation of general or local development; or atrophies. Thus there may be slowness of general growth, or of acquirement of motor function, dental defects and developmental anomalies, also atrophic dystrophy of reproductive organs, or of bone, or of brain (backward children, imbeciles); or even an arrest of development in a permanently infantile, or in an almost mindless state.

(*d*) Morbid predispositions to disease of the nervous system (*e.g.*, to convulsion, meningitis); or to osseous scrufulo-tuberculous affection (Pott's disease: *morbus coxæ*); perhaps even to tubercular lupus.

Besides these organic states also are para-syphilitic changes of anatomical, or of dynamic order; alleged examples of the former being rickets, hydrocephaly, meningitis, congenital spasmodic tabes (Little's disease), the juvenile form of tabes dorsalis and of general paralysis; and examples of the latter, or *dynamic* changes, being neurasthenia, hysteria and epilepsy; these last being somewhat doubtfully offered for acceptance.

Several forms of *familial* disease have also been considered parasyphilitic. For example, recently were published four examples, *in a family*, of infantile spastic cerebral diplegia, attributed to parental syphilis.

We need not again describe, having already considered, the morbid anatomy (when any) of para-syphilitic diseases: how they are without syphilitic gumma or gummy infiltration: how lesions, if present, are those common to many slow long-lasting infectious maladies, and consist of forms of vasculitis, of sclerosis, and of degeneration, usually more or less disseminated, and how, when grave palpable lesions are absent, the dynamic or functional defects and disorders (neurasthenia, hysteria, epilepsy, &c.), are engendered by what I have termed *syphilaemia* (with its structural, toxic, anæmic, chlorotic elements), and by the perturbing mental effects incidental to knowledge of the acquirement of syphilis, and the tragic drama, present and portending, of syphilised life. And, in cases, these factors may be reinforced by ill-effects on nervous nutrition wrought by syphilitic alteration of blood-vessels; or by the mercurialism of over-dosage.

V.—Para-Syphilitic Epilepsy.

The fifth part of my previous communication was on "Syphilitic Epilepsy," so-called. Here may be mentioned the form described by Fournier, as "Para-syphilitic Epilepsy."

Para-syphilitic epilepsy is sudden, unexpected, spontaneous in invasion, comes on usually in an advanced stage of syphilis; takes the form of the great or little epilepsy—the convulsive or vertiginous—is, or may be, isolated; does not yield to specific anti-luetic treatment, yet may be abated by bromide of potassium.

Let us expand this summary a little, partly in Fournier's own words, and entirely on his lines:—

Occasionally, in syphilis is an epilepsy which is very different from "syphilitic epilepsy" or more correctly, from "the epileptic form of cerebral syphilis." It is especially distinguished by the following features:—

It is produced, begins and continues throughout like epilepsy, pure and simple, as an isolated symptom, that is to say, without the accompaniment of other phenomena, and particularly without cerebral phenomena.

It is persistent and tedious in its duration.

It does not yield to anti-syphilitic or other remedies, yet may be controlled, though not cured, by bromides.

The invasion of para-syphilitic epilepsy is no less sudden than unexpected, and apparently spontaneous. It begins as a severe attack of convulsions (*grand mal*), or as a succession of attacks of *pétit mal*.

The attacks of *grand mal* are relatively few and far between; those of *pétit mal*, on the contrary, are relatively very frequent.

The seizures of *grand mal* reproduce all the features of a classical epileptic fit, so also as regards the *pétit mal*, and that of ordinary epilepsy, "apart from differences of detail."

This epilepsy is pronouncedly persistent and of long duration.

It belongs to the tertiary stage of syphilis, of which it is a late manifestation. It begins, as a rule, between the ages of 37 and 48.

The derivation of this epilepsy from syphilis is proved: (1) by indubitable syphilitic antecedents; (2) by the age at which the comitial symptoms first manifest themselves; (3) by the absence of any assignable cause, other than the syphilis; (4) by the occasional birth, to such parent, of an emaciated, degenerate, badly developed child, such as we so constantly associate with hereditary syphilis.

The convulsive form of para-syphilitic epilepsy never assumes the circumscribed and partially convulsive epileptiform type, it is invariably generalised. Para-syphilitic epilepsy continues pure and simple without admixture of other cerebral phenomena, *e.g.*, congestive, intellectual or motor, such as occurs in "syphilitic epilepsy." It persists in its original form for years. It is not amenable to treatment by Hg. and I.

A syphilitic lesion of the encephalon is unlikely as its origin. It comes from syphilitic source, has no features which pertain really to syphilis, or which are solely and exclusively syphilitic; is not amenable to anti-leutic treatment, and by conjunction of all these tokens it is a *para-syphilitic affection.*

VI.—Functional Nervous Diseases and Syphilis. Functional Nervous Disease of Syphilitic Origin.

In the preceding section on para-syphilitic or meta-syphilitic nervous diseases we have partially dealt with the present subject; and under the functional division of these affections paid especial attention to neurasthenia, hysteria and epilepsy.

Paul Kowalewsky arranges the chemical changes of nervous tissue elements as being conditioned by syphilis in five ways:—

I.—By blood changes.

II.—By heroic anti-luetic treatment.

III.—By pathological change of blood-vessels in syphilis.

IV.—By mental shock due to the fact of infection by syphilis.

V.—By the chronic action of the syphilitic virus or toxine.

Taking these in order, the following brief summary of his statements will suffice here:—

(1) Alterations of the blood in syphilis.

Hyper-albuminosis and oligocythaemia were among the earliest-described of the blood-changes. The blood-changes in syphilis are sometimes mainly of anæmic, sometimes of chlorotic type, and, whether primarily due to syphilis or to its treatment, there may be red cells over-big or undersized, or pale and wan; or aberrant in shape, or even strangely deformed. The white corpuscles with single nuclei have been found over-abundant; those of the multi-nuclear variety decimated.

In early syphilis, eosinophile cells have been reported much augmented in number. Hematoblasts and Bizzozero's platelets have been observed to lessen in number during the eruptive and chlorotic periods; and, subsequently, to rise again. The red corpuscles and multi-nuclear white have been stated, by Anz, to follow parallel lines of change; but rising and falling in number inversely as the uninuclear elements, and transition forms, especially eosinophiles.

Roughly speaking, these blood-changes are proportionate to the intensity of the syphilitic phenomena; return with luetic relapses; are influenced by anti-syphilitic treatment; and in a happy issue of the fundamental malady disappear for good, effaced by a gradual return to the normal standard of blood-composition and structure.

These hæmic changes reaching their height, as a rule, coincidentally with the *acmé* of the secondary stage and its syphilitic toxæmia, the tendency is to reappearance of the normal standard; although the hæmoglobin remains somewhat low.

Naturally, these morbid hæmic states lead to defective nutrition of nervous system, to easily produced functional nervous disorder, especially in those otherwise predisposed; and this holds good both in acquired and in hereditary syphilis.

Taking now, and very briefly, the other several points mentioned by Kowalewsky :—

(2) The production of functional nervous disorder through heroic anti-luetic treatment (Hg., I.), is a result of inanition, or of poisoning, not syphilitic, but by an inorganic chemical substance; and we pass it by as being somewhat out of place.

(3) Altered vessel-walls occasion many symptoms in leading towards some of the worst forms of brain syphilis. But irrespectively of this, the irregularities, defects and inequalities of nutrient supply, and of molecular excretion, obviously act so as to foster defective nutrition of nervous elements, and hence promote nervous disorder.

(4) Knowledge, on the patient's part, that syphilis has been incurred, may strike home, producing mental shock; which is obviously in line with like mental factors which do, notoriously,

produce or aggravate various nervous and mental functional disorders.

Applying these views to syphilitic *neurasthenia*, Kowalewsky divides into several forms of it :—

One is the congenital form of syphilitic neurasthenia.

Another is that acquired during the dyscrasia of syphilis.

A third is the inanition-neurasthenia due to mercurial overdosing for syphilis.

The fourth is that occasioned by mental shock in consequence of the patient becoming aware of his luetic infection and of its consequences.

Also as regards *hysteria* in syphilitics, are several varieties; namely :—

(*a*) The form due to intoxication of nervous elements by syphilitic poison.

(*b*) That originating in altered nutrition of nervous elements under influence of syphilitic dyscrasia.

(*c*) That springing from the mental perturbations aroused by the fact of the acquisition of syphilis, and its effects.

(*d*) That brought about by the mercurialism of antiluetic treatment.

Moreover, syphilis in the ancestry may conduce to hysteria in the descendants; an inherited state disposing to hysteria may have its rootlets in ancestral syphilis, and the hereditary degeneration springing therefrom.

Other forms of functional disorder are, in cases, due to syphilis, such as chorea, and angina pectoris.

The examples of *chorea* in acquired syphilis are but few; it is mainly *hereditary* syphilis which is potent in making for the chorea of Sydenham. And the probability is that those seemingly occasioned by acquired syphilis are in reality hysteria—are exemplars of hysterical mimicry of chorea originating in conditions the outcome of syphilis.

In the production of *angina pectoris*, Kowalewsky ascribes several methods to the *rôle* of syphilis :—

One is by the production of morbid changes in the heart-walls :—

Another by making pathological disorder of the vessels :—

And a third by change affecting the cardiac nervous apparatus.

Where the angina pectoris is from syphilitic alteration of the cardiac nerves we find nearly pure angina pectoris; of secondary grade is it in the cases of syphilitic change of cardio-muscular and connective tissue; and still more insignificant is the purely anginal character with syphilitic change of the vessels.

Anti-luetic treatment has been reported very successful in several cases. The happy issue of that treatment would tend to indicate a form of angina pectoris directly the outcome of active syphilis, and not of para-syphilitic nature.

It has often been noticed that, at the outset, organic syphilitic and other diseases of the nervous system are apt to be confounded with functional disorder. Also recently it has been pointed out by Stieglitz that, in the early stage, syphilis of the brain is most frequently confounded with the functional neuroses; neurasthenia, hysteria, epilepsy. This is owing to the transitory character of many of the focal symptoms, and the frequent presence of such neurasthenic symptoms as insomnia, general irritability and headache. One of the cases lately published by C. K. Mills was an example of the kind; at first supposed to be one of hysteria or neurasthenia, but turning out to be brain syphilis in reality. And not a few resemble his third case—with mental weakness, convulsions, polyuria. Deutsch also is alive to the frequency with which nervous symptoms are among the first indicating syphilis to have become a constitutional disease—in other words, the infection to have become general. Such symptoms are pains in head or kidneys; mental symptoms; pallor and nausea (from meningeal irritation). Occasionally, the symptoms of encephalic origin are sluggishness or inequality of pupils, neuralgia, greediness, excessive thirst, severe sweats; and, as for the spinal cord, increased reflex excitability of skin and tendons, followed by rapid lessening or loss of the same for several weeks, even after the rashes have disappeared.

Somewhat contrary to the preceding views, are those of Orchansky, who contends for the existence of a sort of antagonism beween syphilis of the nervous system and neuropathic heredity. He declares that nervous affections are more rife in those syphilitic persons who are without, than in those of them with, morbid neurotic inheritance. Moreover, that this inheritance ameliorates the effect of syphilis by tending to replace organic (gross) nervous disease by functional. And furthermore, that in the production of morbid conditions of the nervous system, syphilis acts almost solely as a factor of organic disease and not of functional.

(*To be continued.*)

Reviews and Abstracts.

Hallucinations and Illusions. A Study of the Fallacies of Perception. By EDMUND PARISH, 1897. "The Contemporary Science Series."

THIS is an English edition of an earlier German work, published at Munich in 1894. The author has recast some of the chapters, dealing with "Waking Hallucinations," and has added a new chapter indicating the relation of the views set forth, to psychology in general.

The book consists of ten chapters, with an appendix containing narratives of "Waking Hallucinations" collected by Baron von Schrenck-Notzig, and classified tables of the same. There is also a copious index and bibliography.

Chapter I. deals with universal fallacies of perception, experienced normally and necessarily by all persons. Instances of such fallacies are the spectre of the Brocken, the illusion of "a straight stick bent in a pool," and the like. Others depend on "ambiguity of the stimuli," for instance, the well known optical illusions produced by such pictures as that of Christ on St. Veronica's handkerchief, or the Death's head, which resolves itself on close inspection, into two girls playing at a window.

Ambiguous stimuli may also depend on the percipient himself, *e.g.*, odours may be confused by one who is not skilled in making subtle olfactory distinctions.

Sensory delusions, such as the sensation that two peas are present when one is rolled beneath the crossed fore and middle fingers are well known. Pathological disturbance of the organism accounts for many other sensory delusions, such as xanthopsia in santonin poisoning, icterus and enteric fever. The sensations referred to an amputated limb are also mentioned in the category of "eccentric projection" of sensation. The author insists that there is no essential distinction between such normal fallacies of perception common to all, and those which occur in morbid conditions of the subject.

Can a distinction be drawn between actually seeing, hearing and perceiving things, and only believing that one sees, hears, or perceives them? He answers that to "believe one sees" and "to see" are two expressions meaning the same thing. It is, therefore, unjustifiable to regard hallucinations as ideation pure and simple. "All hallucinations and illusions may be reckoned as fallacious perceptions whether observed in the sane or insane, whether occurring in sleep, or in the waking state, whether arising spontaneously or experimentally induced."

Hence the author draws no hard and fast line between hallucinations and illusions, as Esquirol did, when he defined illusions as "false interpretations of external objects"; hallucinations as "subjective sensory images," which arise without the aid of external stimuli, but are projected outwards, and thus assume apparent reality.

"Such a definition" he says, "is open to the reproach of employing a physical differentia in a matter purely psychical."

Hallucinations and illusions are alike, fallacious perceptions—whether or not there is an object to be perceived.

Before giving his own points of distinction between hallucinations and illusions, he deals in chapter ii. with "fallacious perception in various pathological and physiological states, in amentia, dementia, melancholia, mania, 'folie circulaire,' delusional insanity and paranoia, and in general paralysis."

As to the share of the various senses in the false perceptions of the insane, and their significance for the patient, he finds that hallucinations of taste and smell are, on the whole, most infrequent. They are usually disagreeable in character.

Hallucinations of cutaneous sensibility—of the organic sense, and the like, are, as a rule, too vague to influence the content of consciousness directly. They only become of primary significance when a darkened intelligence "seizes upon them as a basis for a new conception of the ego and the environment" (Kræpelin).

The danger of visual delusions for the patient consists chiefly "in the importance they gain for him by confirming his mental delusions." Auditory hallucinations "are, perhaps, fraught with even graver danger for the sufferer, because they lead him to seek for explanations, and these bring delusive ideas in their train . . . and since they are but rarely capable of control."

The various fallacies of perception in psycho-neuroses, such as epilepsy, hysteria, ecstacy; in intoxication by alcohol, chloroform, ether, haschisch, santonin, cinchona, opium, nitrous oxide gas, are enumerated; also those in various somatic diseases, in dreams, in hypnosis and in "crystal visions."

The common characteristic of all these states he regards as *dissociation of consciousness.* "Obstructed association is indicated in almost every case" (p. 71). But he says (p. 73) "it is clear that the state of dissociation is not always the same. Rather we find an endless series of gradations from the deeper stages of beclouded consciousness to one which is hardly to be distinguished from the normal; or, to express it differently, from the slightest indication of obstructed association to its almost complete inhibition, or from the profound cleavage of consciousness, to the mere splitting off of single elements, or small groups of elements. The more complete the obstruction of the association paths, and the deeper the disturbance of consciousness, the more numerous are the sensory delusions (as in collapse and fever delirium, for instance), and the less likely are they to be remembered. Thus, states of profound disturbance of consciousness, like epilepsy and deep sleep, are subjectively described as dreamless; whilst states of only slightly disturbed consciousness, for instance, "the periods of transition between sleeping and waking are regarded as favourable to the occurrence of sensory delusions (hypnogogic and hypnopompic hallucinations). It is natural, therefore, that the occurrence of hallucinations should be reported not only in such transitional states between sleeping and waking, when in bed at night, or during the afternoon siesta, but also in analogous states" (p. 74) . . . "The point illustrated by these cases should, at all events, never be lost sight of in the discussion of hallucinations reported as in the waking state; for if a man imagines that he is awake, he will naturally feel that his actions and conduct are rational, and will, in all good faith, so describe them, but an attentive study of such cases reveals more or less certain indications of dissociation of consciousness" (pp. 75-6). In chapter iii. the indications of "dissociation" in cases of so-called "waking hallucinations" (reported by the S.P.R. in their *Proceedings*, vol. x., Aug., 1894) are given.

Although a large number of the narratives dealt with in the report of the S.P.R. indicate that the informants were firmly convinced that their hallucinations occurred in the waking state, there is frequently evidence that they were mistaken as to their state of consciousness.

In many cases the hallucination was experienced at the moment of waking, or in drowsy mental states, indicated by such frequently occurring expressions as "I had just awakened and given my baby the breast" (p. 94).

Sometimes, especially in "collective hallucinations," there is evidence of suggestion acting in a state of expectancy, when, for instance, a husband sees an apparition, only after his wife has insisted that it is present. In other cases fixation of the eyes or prolonged abstracted gazing on a shining surface, such as a sheet of paper, have had a share in bringing about the phenomenon. In other words a condition of hypnotism has been self-induced. In 62 per cent. of cases the percipient was alone; hence the presence of others—a circumstance which conduces to the waking state, is unfavourable to the occurrence of hallucination.

When the informants state that they "fainted from terror or shock at the apparition," such communications remind us so forcibly of the hallucinations of the epileptic and hystero-epileptic aura, that we can hardly resist the conclusion that they occurred in a semi-conscious state, probably of very short duration, preceding a state of complete unconsciousness" (pp. 99-100).

When there are no indications of disturbance of consciousness, it is doubtful whether we can take the percipient's word that he was fully awake at the time, for granted, seeing that "similar assurances given with the same firm conviction on the part of the narrator proved to be mistaken" (p. 103).

There must often be want of evidence for a state of dissociation, because the narratives (often very meagre) "are occupied mainly with the content of the hallucinations rather than with the state of consciousness which accompanied them." By oft-recital, gradually all subsidiary detail fades away until finally there remain in the memory only two points of cardinal importance—the hallucination itself, and the conviction of having been fully awake at the time—a delusion common with persons in a drowsy, half asleep condition.

Lapse of time destroys the chance of showing from the narrative itself that the phenomena recorded were not hallucinations, but either illusions or objective sensory perceptions mistakenly supposed to be subjective—because subsidiary details are forgotten. It is significant that reports of so-called waking hallucinations increase in number with the number of years which have elapsed. He explains this by supposing that in the older reports all accompanying circumstances which might throw doubt on the genuineness of the hallucination have disappeared, such as, *e.g.*, the state of the light and the physical surroundings, or any indications of a state of dream consciousness on the part of the percipient.

He concludes that waking hallucinations in sane persons are much more frequent phenomena than appears from the tables of the S.P.R., but it is only the more striking ones which remain in the memory and are recorded.

The Physiological Process in Fallacious Perceptions.

In chapter iv. early theories in explanation of the physiological process in false perception are criticised and set aside.

The psychic centrifugal theories "which regard all hallucinations as evoked in consequence of exceptional ideational images penetrating into the region of sense, or in terms of physiology, as the result of a current of centrifugal energy from the cells of the cortex exciting the basal ganglia into activity" (p. 121) are disposed of:—

(1) On the ground that however vivid and energetic an ideational image may be, it can never receive the stamp of sensory reality.

(2) It is difficult to refer to ideational excitation visions which mock at all experience, *e.g.*, visions of a blue dog; but it is easy to connect such an appearance with an illusory perception of a subjective impression of blue light.

(3) If an energetic ideational stimulus could arouse a corresponding activity in the sensory centres, hallucinations would be more common in the sane than they are.

The centrifugal sensorial theories, which refer the seat of hallucinations to the sub-cortical sensory centres, fail because when such centres are destroyed by disease, for instance, "in sub-cortical sensorial aphasia due to disturbance of conduction between the temporal lobes and the centres in the medulla, though word deafness occurs, the acoustic imagination remains intact, and there is no loss of spontaneous speech" (p. 127).

Moreover, we should be chary of assuming a descending current in ascending nerve tracts, since such a hypothesis is inconsistent with generally accepted physiological theories (p. 131). He discards centrifugal psychical theories as well as their opposite or *centripetal theories*, for the reason that "the whole controversy as to whether the process travels centrifugally or centripetally, becomes meaningless when once we have seen adequate ground for concluding that the centres of sensation and imagination are not totally separated but occupy the same part of the brain, and that the difference in character between sensory perception and ideational reproduction corresponds only to a different degree of excitement in the same cells" (p. 134).

Hence the old distinction between hallucinations and illusions will not hold good. Both alike may be reduced to one type—Esquirol's illusions—" All false perception is an anomalous reaction of the brain to sensory stimuli, and the hallucinatory process, only a special form of that process which accompanies all objective perception " (p. 144).

Both hallucinations and illusions are conditioned by what he calls " the dissociative stage." By dissociation he means " that state in which the nerve stimulus no longer flows through the channels determined by habit, and by the co-operation of simultaneous stimuli, because inhibitions, or obstructions, whether from pathological or physiological causes, have been set up in the normal association-paths, or obstructions which normally exist in other connecting tracts have been weakened or altogether abolished " (p. 125). He distinguishes as " illusions," the phenomena which result from the suppression of certain processes, and as " hallucinations " those which are caused by an act of forced association." " No hard and fast line can be drawn between them, though, presumably, either the *plus* or *minus* quality predominates, and the phenomenon can be classed as ' hallucinations ' or ' illusions ' accordingly " (p. 149).

The gist of his argument seems to be as follows :—A is a group of higher cortical centres, which when moved to activity by peripheral stimuli, excites a process " α " which tends to irradiate on all sides in the direction of least resistance, and to discharge certain element groups normally associated with the process α. If such element groups become exhausted, or if the paths normally open to the irradiation of the process α are closed, and if on the other hand a certain close-knit group of elements " β " is in a state of high tension, then when A is stimulated, " α " being able to discharge only in the direction of least resistance, will be forced to discharge towards this group " β," which has perhaps never been affected by it, but only by excitation of another primary group B.

The result on " β " is precisely similar to that which would have been produced by excitation of B.

" The psychical concomitant of this process is a sensory perception, which yet lacks the special sensory stimulus usually associated with it ; in other words, it is a perception without objective basis, a *hallucination* " (p. 148).

On the other hand, supposing the excitability of neural elements to be lowered generally ; stimulation of A may evoke a process α and lead to a perception, although it may fail to start other processes β, γ, δ, which normally combine to produce or modify the general result of a perception. "The psychical concomitant of this brain state is a perception such as 'a' or the sensory process started by a can produce. But it misrepresents the sensation for which it stands because it is incomplete, and lacks the correction and adjustment which the dormant elements in consciousness could alone have supplied. It is in a word an "illusion" (p. 148).

Hence he distinguishes "*as 'illusions' the phenomena which result from the suppression of certain processes, and as 'hallucinations' those which are caused by an act of forced association*" (p. 149).

Chapter V. treats of the factors of fallacious perceptions. The dissociated state may depend on both pathological and physiological causes, which exhaust the elements themselves, and thus produce obstruction to the outflow of energy.

"In psychical concentration a reverse process brings about the same result. The tension in certain selected element groups is heightened, and their irritability increased, but this takes place at the expense of the non-selected elements, when irritability may be reduced to a minimum, causing a state of dissociation or splitting off to ensue" (p. 154).

Dissociation again may be caused by *exclusion* of external sensory stimuli, *e.g.*, in cases of hypnagogic hallucinations, dreams, in the tendency to see ghosts and visions in the dark, and in the fact that auditory hallucinations are nearly always present in the insane deaf.

The action of dissociation is to cause misinterpretation of excitations which normally produce 'correct' or objective sensory perceptions.

The stimulus need not be an objective sensory impression, but may consist in pathological or physiological irritation of the sensory centres. In the normal state both processes are recognised as so-called subjective sensations; but if dissociation obtains they may become causes of false perception (p. 163).

The physiological sensory irritation may depend on changes such as metabolic processes in the centres themselves, and in the nerve tracts leading to them.

The pathological irritation may depend on morbid processes in any part of the brain. Many examples are given of hallucinations, due to excitation of the special senses of vision, audition, of the olfactory, and of the muscular sense.

Cramer ["Die Hallucinationen im Muskelsinn bei Geisteskranken und ihre Bedeutung" (1889),] sought to refer a multitude of phenomena to subjective sensations of the muscular sense. But the objection to his views is that they cannot explain auditory hallucinations. For if the latter be really due to excitation of the muscular sense, the patient must have the sensation that he *is uttering* a certain word, whereas in reality he has no such sensation. In visual hallucinations the patient has the sensation that he *sees*, in auditory hallucinations that he *hears*, and therefore in hallucinations of the muscular sense, he should have the sensation that he *speaks*—but this is not so.

In chapter vi. the "content of fallacious perception" is considered.

This depends on (1) memory and experience. Only what has passed in at the portals of sense can be reproduced. (2) On the conditions which induce the hallucinated state. In the case of disease, the hallucinations are usually unpleasant, in those produced by the action of drugs they will vary with the temperament of the individual. (3) Again, hallucinations will be guided by the environment of the individual, his calling and associates, the beliefs and superstitions incident to his time and country. (4) On the brain state which obtains at the moment (exhaustion, concentration, emotions, sub-conscious processes). (5) On the sensory stimuli. Just as in the dream-state the dream-content depends on sensory stimuli, so the action of these is to be observed in hypnotic and narcotised subjects, in cases of intoxication, in spontaneous somnambulism, and in many hysterical states. Various illustrations are given in point.

The rest of the chapter deals with the phenomena known as "Rapport," in which the hypnotic subject is only influenced by the hypnotist, and exhibits 'negative hallucinations' or non-perceptions of objective sense impressions, which any but the hypnotist attempt to produce.

Rapport and all its symptoms, he refers to "the different degrees of distinctness in the perception according as the elements which the stimuli encounter are in a state of heightened or lowered tension" (p. 208).

If the stimuli encounter elements in a state of high tension, the impressions are perceived hyper-æsthetically, if otherwise they are not perceived at all.

He draws a distinction between the negative phenomena of "Rapport" and true negative hallucinations.

In the former, objects are not perceived because attention is diverted from them. In the latter, attention is directed to the objects, but the direction of the attention is a counter suggestion, that they shall not be perceived.

"Negative hallucinations in contra-distinction to the negative phenomena of Rapport, which have a dissociative character, are conditioned by cerebrostatic enforced association—that is to say, they are true hallucinations in every sense, and the only negative thing about them is the verbal form of the suggestion" (p. 219).

The Initiation of Fallacious Perception.

How is it that sensory stimulation of one sense may produce an hallucinatory response in another? For instance, why should a sleeper dream that he *sees* icebergs and polar bears, when the bed clothes fall from him, and he *feels* the sensation of cold?

He explains such "reflex hallucinations" by "synæsthesia," hat is to say, constant involuntary association of a certain image or (subjective) sensory impression with an actual sensation belonging to another sense.

A particular taste, or smell, or sound, may call up the sensation of a particular colour.

He enters in detail into the phenomena of "colour hearing" (audition colorée) or "sound seeing," and concludes that such hallucinations are due to "an automatic association acquired very early in life" (p. 229).

In the fallacious perception of icebergs, &c., mentioned above, he holds that the temperature stimulus is not the *starting* factor, but merely "co-operates with many other circumstances to bring about the required state of heightened tension in a particular element complex, and thus directs to it the irradiation of processes initiated otherwise by stimulation of the visual sense. It only prepares the way for the hallucination, it exerts only a suggestive influence in its content" (p. 231).

The Manifestation of Fallacious Perception.

Auditory, painful, olfactory and gustatory fallacious perceptions occur in varying degrees of distinctness. Sometimes the hallucinations seem to be scarcely distinguishable from vivid mental images; or again they may be externalised to such a degree as to differ in no particular from the ordinary correct perception of plainly recognised objects.

In *visual* hallucinations there are the same gradations. In the lowest degree there is imperfect externalisation, the percipient sees an image, "in his mind's eye," as he expresses it.

Next, there is true but unsubstantial externalisation. For instance, a dead face is seen on a bed curtain. This form of hallucination corresponds to "after images" induced by prolonged gazing at some object.

The visions are dim, indistinct, shadowy or transparent. In the highest development of visual hallucinations, there are realistic bodily appearances, combined with non-perception of that part of the field of view covered by the apparition.

In hypnotic cases, the vividly externalised phantasms produced by hypnotism may appear even more real than the actual objects which they represent.

The attitude of the percipient with regard to the hallucinatory perception, frequently is not in accordance with what it would be if he believed in the reality of the apparition. But this does not disprove the genuineness of the hallucination.

For instance, in a case quoted by Randinsky, the percipient was not terrified or excited by the apparition of a lion; but "this means no more than that in consequence of the condition of percipient's brain at that particular moment, the complex of elements usually associated with the idea of a lion roaming about at liberty did not and could not take place" (p. 251).

If told that the lion was dangerous, the percipient would have at once evinced alarm.

A medical student was hypnotised by the author, and the presence of a snake was suggested. The subject was not terrified until the operator exclaimed "Look out, it's a rattlesnake." The author assumes that the images were not purely mental ones, but that a hallucination of the senses is to be assumed in both cases (p. 251).

If the behaviour of hallucinated persons cannot be taken as a test, no more can the presence of a certain subjectivity on their part disprove the sensory character of the experience. Some believe that they hear their own thoughts uttered by their own voices; others that their own thoughts are uttered by the voices of others; whilst a third variety believe that their thoughts are not their own, but suggested to them by God or Devil. In all these cases there is a sensory basis for the delusions. "The greater number of 'voices,' if not all, are caused . . . by automatic speech on the part of the percipient" (p. 271).

"Audible thinking," he attributes to auditory perceptions of articulatory movements, which otherwise make no impression on the consciousness.

Automatic writing is another example of the same thing. "Motor impulses set the muscular apparatus in motion while our upper consciousness knows nothing of the action except through the result, afterwards looked upon with incredulity by the writer, who denies having written the words, and either thinks he has been made a fool of or attributes the writing to spirits (mediumistic writing)" (p. 261).

Chapter ix. contains an examination and adverse criticism of the evidence which has been brought forward in favour of telepathy.

It has been urged that the frequency with which apparitions coinciding with the death, or with some exceptional crisis in the life of the person whose presence they suggest occur, is too great to be merely fortuitous.

But he maintains that the evidence of the coincidental and veridical[1] character of the phenomena is by no means satisfactory. In many reported cases there is obvious indication of the tendency to connect events, especially those which are important and striking, with each other. For instance, a man is awakened by a noise which he cannot explain. Three weeks later his father-in-law dies. He therefore assumes that the noise and the death are somehow related to each other.

Hallucinations of memory, particularly the impression that one has expected some exciting experience long before it actually occurs, will account for another class of cases. "Present impressions are projected backwards in a form involuntarily modified to suit the presentment" (p. 278).

Also Kræpelin's "identifying fallacy," in which there is a sense of "banality," or of having passed through the same experience before its present occurrence, may explain other so-called presentiments.

Sometimes an actual hallucination is adapted to an event afterwards connected with it. "The points of correspondence come out with special distinctness; those, on the contrary, which do not correspond drop out of sight or are assimilated to the rest" (p. 283).

Coincidences are not always proved. The percipient of an apparition transfers the hallucination to the day of the subject's death, and not the death to the day of the hallucination.

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[1] By "veridical" he means "depending in some way on the event which may shadow forth" (p. 272).

The most important flaw in the evidence that hallucinations of persons dying appear coincidentally to their friends—evidence which if strong, would be greatly in favour of telepathy—is that the instances recorded as occurring more than ten years ago, are nine times more numerous than those occurring within the last five years (p. 289). This fact alone is sufficient to shake faith in the accuracy of the deponent's statements. The more recent cases usually break down on cross-examination.

But even if coincidences between impressions and events be proved, "they afford not even the shadow of a proof for telepathy" (p. 301). A believer in telepathy feeling cold, may "wish" that a friend, far away, should also feel cold, but even if the friend does feel cold coincidentally, it is no proof that telepathy is at work. The friend's window may be open, or his fire out, or his bedclothes may have fallen from him.

In conclusion, he gives many instances of 'dissociation' in the death-coincidences of the Report of the S.P.R. "Dissociation of consciousness" is the keynote of the book. By evidence of its occurrence he seeks to explain all the illusions and hallucinations of the sane and the insane, of the healthy and of the diseased, whether asleep, or believing themselves to be awake, whether arising spontaneously or experimentally induced.

LEONARD GUTHRIE, M.D.

Anatomical and Physiological Observations on the Organ of Vision for the Purpose of Ascertaining the Alteration caused by Section of the Optic Nerve.—By Augustus Waller, M.D., F.R.S. Part 1, received December 10, 1855, Read January 17, 1856.

[Extracts from an unpublished paper in the Archives of the Royal Society. The paper bears evident signs of incompleteness; it is entitled Part I., and concludes with the sentence:—"In the following part of this paper I intend to describe my experiments in tracing the fibres of the optic tracts into the deeper parts of the brain, and the results obtained from section of both optic nerves." This intention was not fulfilled, but the extracts, as they stand in the words of their author, possess historical interest, and very clearly prove that Waller was equally alive to the principle established by subsequent investigators, that the retinal ganglion cells control the nutrition of optic nerve fibres, and to the application of that principle in the tracing out of optic paths. An abstract of the paper is contained in the Proceedings of the Royal Society for 1856. A point of detail of some interest is the view taken by Waller of the chiasma in the rabbit; he thought the decussation was complete, although expressing himself with some reserve. Von Gudden (who in Germany and in England is generally quoted as the originator of the degeneration method as applied to the retina and optic nerve) went through a similar difficulty in connection with the rabbit's chiasma in 1874, concluding at first that the decussation is complete, but subsequently detecting the relatively small uncrossed tract of degeneration that proves the decussation to be incomplete, in the rabbit as in the dog, monkey and man.—A. D. W.]

THE following observations, which I have the honour of submitting to the Royal Society, confirm the results of an inquiry undertaken for the purpose of ascertaining the alterations which ensue in the living body after division of the optic nerve. This mode of investigating the nervous system was first communicated to the Royal Society in a paper which the Society did me the honour of publishing in their Transactions; since which I have had to put in practice this mode of inquiry, which has been the means of elucidating several important facts in reference to the nervous system.

SUMMARY OF OBSERVATIONS ON THE SPINAL GANGLIA.

The necessary deduction from these observations is that the spinal ganglion performs the office of sustaining the nutrition of the nerve fibres with which it is connected. In other words, it may be concluded from these experiments that the spinal ganglion is the centre of the nutritive power of the nerve fibres adherent to it. Experiments on the spinal cord showed that the anterior spinal roots derived their nutritive influence from the cord. On account of the general nature of the phenomena in question, and of the fact that nerve fibres are always connected with a centre of grey matter, little doubt can remain that every nerve fibre is in connection with a nerve cell, which exerts over it a peculiar influence, enabling it to retain its structure, and by so doing to serve as the highway for motor and sensory power to and from the brain.

It would, however, be an undue generalisation to conclude that all ganglia exert the same influence as those of the posterior spinal roots whose influence is of a bipolar nature; and, indeed, similar experiments have shown, on the course of the ramifications of the sympathetic nerve, that certain ganglia, such as the first cervical sympathetic, have only an unipolar influence on their dependent nerve fibres.

What remains, therefore, to be done by means of this process, is to *determine experimentally for each nerve, or set of nerve fibres*, the condition of the two ends of a nerve which has been divided on an animal which has continued to live for a certain lapse of time after the experiment. The nerve which I have at present under consideration is the optic nerve.

Different observers, nearly all equally trustworthy from their high position in science, have been cited to prove in turn that all the fibres of one eye are derived from the opposite side of the brain; that none of them are derived from that source, but from the same side of the brain; and, finally, other cases are adduced to prove that at the chiasma a mingling of the fibres from opposite sides takes place.

By means of the process now available it will be endeavoured to clear up some of the causes of error and to place this inquiry on a more solid basis.

Structure of the Optic Nerve. Method of Operation. Effects of Division of the Optic Nerve.

These may be studied on the retina by means of the ophthalmoscope during life, and on the dead animal by microscopic examination of the retina and of the peripheric portion of the optic nerve connected with it, and of the cerebral portion of the nerve from the divided end to the quadrigeminate bodies. In the eye of the rabbit the optic nerve sub-divides into two portions of nearly equal size, which spread out into two wing-like expansions over the internal surface of the choroid, forming a most striking feature by their brilliant white colour, reflecting the light much more strongly than the retina which surrounds them. As the optic nerve enters the eye at the upper and outer part of its optic axis, this bright portion of the retina can only be easily perceived when the pupil is fully dilated. Repeated inspection of this structure at various intervals after section of the nerve, varying from two or three days up to as many months, have not enabled me to detect any alteration in the aspect of this part of the retina. Under the microscope this expansion of the optic nerve consists of tubular fibres, beautifully clear and defined, and like those composing the optic tract, except that they are smaller; the diverging filaments visible to the naked eye consist of fasciculi of these fibres, which before being blended with the grey fibres of the retina, are not unfrequently seen to split up into exceedingly fine filaments, which generally arise from a varicose dilatation of the tube. I have examined these fibres at various periods after section of the optic nerve—at first only a few days after the operation ; in the other cases at the end of a month and upwards to four months, and in every instance have found them to be as clear and as perfect as in the opposite and sound eye. The elementary parts of the retina likewise appeared unaltered, as well as the parts composing Jacob's membrane. My observations on the peripheric end of the optic nerve, or that in connection with the eye, have been less numerous than those upon the retina, as this part of the nerve was generally removed in order to prevent the reunion of the divided ends of the nerve. I have, however, observed this portion about a month after section, when its fibres were perfectly sound, as well as those in the retinæ; while the cerebral part of the nerve in front of and behind the chiasma, throughout the whole extent of the root, consisted of fibres nearly all disorganised and reduced to a granular condition.

In the case of a rabbit, about three months after section of the right nerve, the fibres anterior to the chiasma appeared all disorganised. The various fasciculi of fine fibres, of which the nerve on the sound (left) side consisted, had nearly disappeared on the cut side, and in their place was found a tissue composed of granules and globules, like those of fat dispersed in the membranous parts of the nerve which presented very slight traces of its normal fascicular structure. At the posterior surface of the chiasma, their superficial sections, made nearly parallel with its surface, presented on the cut side (right) the ordinary appearance of disorganised fibres, which quickly assumed an oblique direction towards the left optic root, crossing nearly at right angles the sound fibres proceeding from the sound nerve on the opposite side.

In the chiasma the distinction between the sound and the diseased fibres was very manifest, and the manner in which each set of tubes crossed from one side to the other was beautifully displayed by removing thin slices from the posterior surface of the chiasma. From the right side the disorganised fibres generally appeared to run in bundles, which were intersected by others of sound fibres, sometimes on the same plane, sometimes by others deeper seated. When the disorganisation is far advanced, as in the case above described, single fibres, or very fine fasciculi, composed of few fibres, could not well be seen from the resorption of much of the granular matter, but the sound fibres from the other side mixed with granular matter, were found running one or two together and mixing with the other in every variable combination. The plexiform arrangement, therefore, of the optic nerves may be regarded as complete in the chiasma, whether as regards the fasciculi of the nerves or their elementary parts. In the optic tract or roots, probably on account of the greater size of the nerve fibres which compose them, the alterations resulting from degeneration of the nerve are still more evident than in the chiasma and the nerve in front of it. It is then found to contain compound cells of black grains, spherical cells nearly colourless, fine granular matter and normal fibres. The compound bodies were generally globular, but sometimes considerably elongated; the first measuring about 0.015 mm., the latter sometimes reaching 0.024 mm. The black grains were of various sizes, varying from 0.002 mm. to 0.020 mm. The larger grains were rather transparent and nearly globular. The transparent globules, of about the same dimensions as the larger grains found in the compound cells, were rather yellow and destitute of any nucleus, resembling in every respect globules of a fatty nature. Granular matter, such as existed in the chiasma and other parts of the nerve, existed in abundance at every point of the disorganised nerve, distributed either in lines or irregularly dispersed. Generally, on account of the increased opacity of the nerve resulting from these alterations, I found it advisable to submit the thin slices removed from it to the action of caustic soda, in order to render them more transparent. Among these diseased elements were to be seen several transparent varicose nerve tubes, perfectly normal to all appearance. The plum-pudding-like appearance assumed by these different elements embedded in the nerve, is different from that of any of the disorganised nerves that I have hitherto examined. The compound granular bodies which give this structure its peculiar appearance, can at present be only referred to the varicose swellings, which at this point are found so frequently and are so considerable in this part of the nerve. The other disorganised matter closely resembles what is found in other disorganised nerves; and if in this place they are apparently extravasated from the tubular sheath, it is probably owing to the greater tenuity of the latter, which has rendered it more easily removable by absorption.

In the optic tract of the same side as the section, I have not been able to perceive any altered fibres. As some physiologists have been of opinion that different kinds of fibres entered into the composition of the optic nerves, some running from one retina to the other, others from the quadrigeminate bodies of one side to those of the other, and, finally, some fibres direct from the quadrigeminate bodies of one side to the corresponding eye, it may not be considered uninteresting to weigh the evidence on these points to be obtained by disorganising the nerves. With regard to the fibres believed to unite the two retinæ, if they are supposed to have two nutritive centres in the nerve cells of each eye (an hypothesis which my researches on this subject do not allow me to admit), we should find in the divided nerve some sound fibres on the superficies over the inner part of its surface still nourished by the sound eye, such fibres I have not hitherto been able to detect. If we admit that each eye is the nutritive centre of half of these direct fibres, we ought to find a mixture of sound and altered fibres in the nerve which has been divided, and in that which has been left untouched, an arrangement which I have not detected. With regard to the fibres connecting the quadrigeminate bodies, on examining the posterior rim of the optic tract on the side opposite the section (left side), it was perceived to be formed of fibres all apparently sound, although the rest of the tract was disorganised. The corresponding rim of the right tract was likewise quite sound. The presence of fibres connecting the quadrigeminate bodies may therefore be regarded as probable.

Direct fibres between each eye, and the quadrigeminate bodies running along the external rim of the tract, would require the presence of disorganised fibres in the external rim on the same side as the section. Such I have not found to have been the case in the instances where I have examined these nerves. On the same side as the divided nerve the external rim of the tract was perfectly sound, on the opposite side the external rim consisted of disorganised fibres, which seemed to contain some sound ones. With regard to the normal fibres found in the tract among those diseased, it would be at present premature to come to any conclusion respecting their origin without fresh experiments, and the examination of those tracts after the section of both optic nerves.

The fibres of the optic tract may be traced with great facility over the surface of the internal or anterior geniculate body, where they present the appearances described in the optic tract. Their course in the external geniculate and quadrigeminate bodies will be described in the succeeding part of this paper.

The most important result of the foregoing observations is, that a great part, if not all, of the fibres of the optic nerve have their nutritive centres in the retina, and from the analogy existing between the nerve cells of the retina and those of the spinal ganglia we are led to attribute this nutritive power to them.

In the following part of this paper I intend to describe my experiments in tracing the fibres of the optic tract into the deeper parts of the brain, and the results obtained from section of the optic nerves.

No published record exists of the experiments last referred to, nor have I discovered any memoranda relating to them among my father's papers.—A.D.W.

Sachs on Multiple Sclerosis. (A Critical Digest.) *Journal of Nervous Diseases*, 1898.

Multiple or disseminated cerebro-spinal sclerosis has for many years been considered to be one of the most easily recognisable diseases of the central nervous system. Under the leadership of Cruveilhier and of Charcot, the French school described the disease so accurately that there appeared to be little to add to the accounts as given by them. The typical forms are indeed unmistakable, but the disease appears so often under all sorts of disguises that its recognition may be by no means easy. Moreover, the interest in it is not exhausted by the establishment of the diagnosis, for it has important bearings to other diseases, and its etiology and pathology present many points for further elucidation. In America the disease is not as frequent as in France and in Germany. The time seemed ripe, therefore, for a consideration of the disease; but we will refer only to the more important questions that have been raised regarding it during the last few years, as the work of preceding years has been well summarised in the publications of Charcot, Marie, Gowers, Oppenheim, Dana, Pritchard, and Gray.

Clinical Symptoms.—The following table of symptoms, as given by Charcot, may be taken as a starting-point for the consideration of the symptomatology of multiple sclerosis. All the symptoms are rarely present together, and, according to the predominance of the several series of the disease, assumes spinal, cerebral, and cerebro-spinal types.

I. SPINAL SYMPTOMS.

Positive.	Tremor on voluntary movements of the extremities —"Intention tremor" (arms and head; more rarely of legs). Titubation. Paresis (spasmodic) of the extremities. Contracture, with exaggeration of the reflexes—spastic rigidity.
Negative.	No sensory symptoms, or only very slight disturbance. Vesicle disturbance, none or very slight.

II. CEREBRAL SYMPTOMS.

Dysarthria—slowness of speech; scanning of words.
Nystagmus—blank expression.
Attacks of vertigo—spasmodic myosis.
Transitory amblyopia—white atrophy of the papillæ.
Diplopia—associated paralysis of ocular muscles.
Mental enfeeblement.
Apoplectiform and epileptiform attacks.
Difficulty in deglutition.

III. ABNORMAL OR UNUSUAL SYMPTOMS.

Trophic.	Muscular atrophies (amyotrophies), bedsores.
Tabetic.	Lightning pains. Romberg symptom. Anæsthetic areas. Vesicle and rectal paresis. Gastric crises.

Frequent remission of all the symptoms.

Charcot recognised three distinct periods in the development of the disease:

First period. The disease may begin with cerebral symptoms (attacks of vertigo, transitory diplopia, scanning speech, and nystagmus); more frequently the spinal symptoms are the first (a slowly progressive paresis to which intention tremor is soon added); in rare instances there is an apoplectic attack, preceded by vertigo and headaches, and followed by hemiplegia. The rarest of all onsets is the one with gastric crises, which are followed by other symptoms. During the second period the typical symptoms are developed, and after years contractures set in. The third period is characterised by mental enfeeblement, almost unintelligible speech, great physical weakness; finally, vesical troubles, with subsequent cystitis, bedsores, pyæmic conditions—death. Babinski claimed that an ascending acute myelitis was an occasional cause of death.

Marie has insisted upon a division into four different types, according to the mode of progression: (1) The chronic progressive type—a very gradual increase in all the symptoms, and covering a period of years. (2) The chronic type, with exacerbations, marked by the occurrence of hæmorrhages, sudden amblyopia, and the like. (3) The chronic remitting type—the progress is very slow, with only slight exacerbations from time to time. (4) The type characterised by permanent improvement (and even cure ?). Almost all of the chronic disorders of the nervous system, tabes, for instance, could be subdivided in the same way. These divisions do not appear to be distinctive. To Charcot we owe the recognition of imperfect forms of multiple sclerosis, in which the symptoms that have existed have disappeared. In these abortive forms (*formes frustes*) some of the important symptoms are never developed, and in others the symptomatology is obscured by the occurrence of hemiplegic, tabic, or amyotrophic symptoms. Oppenheim has at various times directed special attention to the variability of the symptoms, the apoplectiform and vertiginous attacks, and to fundus lesions without functional defect.

The preceding statement is sufficient to show the extreme variability of the clinical pictures as a whole, and explains the possibilities of mistaken diagnosis. During the past few years some of the special symptoms enumerated in Charcot's table have received further elucidation. Strümpell maintained that the intention tremor of multiple sclerosis differs little, if at all, from ordinary ataxia. The author has at present under observation three undoubted cases of multiple sclerosis, whose movements are distinctly ataxic. But in other patients suffering from multiple sclerosis, the disturbance of motion is more of the nature of a tremor. A typical tremor is often present at the beginning of the disease, and as the disease progresses the tremor becomes coarser, resembling ataxic movements.

Gowers in a foot-note (vol. ii., p. 548) states that *ataxia tremor* is the best translation for "Intentions-Zittern"; he chose the term Ataxic Tremor, "because there is no ataxia without voluntary movements." The difference between ataxia and tremor—a difference in degree only—can be brought out by asking the patient to write with chalk or lead pencil, when it will be found that the ataxic patient scarcely succeeds in the attempt to write, while the patient with tremor writes with some difficulty and in in a jerky fashion.

The Ocular Symptoms have been studied by Parinaud, who reported upon the findings in Charcot's cases, and by Uhthoff, who described with great detail the clinical and anatomical findings in the eyes of patients suffering from multiple sclerosis. Recent contributions to this special subject have been made by

Luebbers—a pupil of Uhthoff—by Guenther, Nagel, Kunn and Schwarz.

The commonest symptoms are the nystagmus and the nystagmus-like movements. These are most apt to appear on extreme lateral movements of the eyes, and are generally associated in character. While occasional twitching movements of the ocular muscles may occur in healthy persons, regularly developed oscillatory movements upon each lateral excursion are characteristic of the disease, but of course it should be borne in mind that the symptom is present in other diseases of the nervous system.

Kunn described a condition which he calls a "fixation tremor" setting in when the eyes are moved from vision straight ahead to the fixation of a definite object. The same author calls attention to a concomitant strabismus which he interprets to be a dissociation of ocular movements that were previously associated.

Luebbers mentions an exceptional case in which the nystagmus appeared with converging movements of the eye. The nystagmus may be horizontal, vertical, diagonal or rotary. Raehlman and Schwarz dwell upon the special form of ataxia nystagmus, which is practically an uncertainty of movement, the eyes simply over-leaping the mark in passing from one point of fixation to another. The same phenomenon is observed in Friedreich's disease, and can be distinguished from the more rapid movements of typical nystagmus. True nystagmus occurs in a majority of cases of multiple sclerosis (according to Uhthoff, in 58 per cent., and according to Marie, in 70 per cent. of all the cases). The variations in the percentages are accounted for by the fact that Uhthoff draws a distinction between nystagmus and nystagmoid movements. These oscillatory movements of the eyes constitute, therefore, one of the most constant and reliable signs of multiple sclerosis. They occur, however, in different forms of congenital defective development (idiocy, hereditary spastic paraplegia, etc.), also in Friedreich's disease, in hereditary cerebellar ataxia, and in a few cases of syringo-myelia. This fact is responsible for much of the confusion that has arisen in the attempt to differentiate between these states.

Uhthoff states that affections of the optic nerve are frequently associated with true nystagmus in multiple sclerosis, whereas nystagmus alone, without such atrophy, occurs in other conditions.

The cause of nystagmus according to Uhthoff is due to lesions in the brain, and particularly in the medulla oblongata;

but may be due to changes in the peripheral nerves. A lesion involving the posterior longitudinal fasciculus was supposed by some to cause nystagmus; but, as Redlich has pointed out, this tract has been entirely exempt in cases of multiple sclerosis with marked nystagmus, and in some cases in which nystagmus was absent, this same bundle was seriously affected. In all probability, any lesion interfering, by lesions of the nuclei or of the associating tracts, with the regular movements of the ocular muscles, would be sufficient to account for nystagmus.

Paralysis of the ocular muscles occurs in multiple sclerosis. It has been present in three of ten cases which have come under Sachs' recent observation. The abducens is most commonly affected. Next in order of frequency comes the oculo-motor nerve, which is, however, rarely affected in its entirety. Occasionally the trochlear (IV.) nerve is involved. A point of some importance is that these palsies are variable, disappearing in some weeks or months, and occasionally returning again to as marked a degree as before. The remission in this one symptom is as characteristic of multiple sclerosis as the remission in most of the other symptoms, and it is the occurrence of such remissions that makes it particularly difficult to distinguish between disseminated sclerosis and multiple cerebro-spinal syphilis (Oppenheim, Sachs).

Paresis or paralysis of associated movements also occurs. The upward and downward movements are, as a rule, not affected (Parinaud, Marie, Uhthoff, and Luebbers). Parinaud claimed that paralysis of convergence was frequent (Uhthoff says in only 3 per cent.). A complete ophthalmoplegia externa has not been observed. The direct cause of ocular palsies is supposed to be the sclerotic patches occupying the region of the various ocular nuclei. The palsies do not appear, as a rule, until long after unmistakable symptoms of the disease have appeared. The pupils may be unequal, and sometimes react sluggishly to light and during accommodation, but iridoplegia is very rare. Charcot insisted upon the great value of this preserved light reflex in diagnosis. Damtsch says that the condition of hippus is often found in multiple sclerosis.

Much more characteristic are the changes in the visual power, in the visual fields, and in the structure of the papillæ and the optic nerves. The subjective disturbances are much less than the objective changes. Complete blindness is very rare, although transitory blindness has been observed (Schwartz). Complete recovery from the visual defect of multiple sclerosis is possible. According to Uhthoff, Luebbers and others, central scotomata are most frequent. In this area the colour sense is the one most disturbed—generally for red and green, sometimes for all colours. These central anæsthetic areas may occur together with the normal peripheral fields, or with a mere diminution of peripheral vision. In some cases there may be a peripheral contraction of the visual field with normal central vision. Concentric limitation of the fields has been described by Oppenheim, Thomsen, Buzzard and Uhthoff, but only in cases in which an hysterical or a functional condition was superimposed upon disseminated sclerosis. There is usually a disproportion between the visual defect and the ophthalmoscopic appearances. In one of Luebbers' cases the visual defect was in keeping with the changes in the papilla, but in two others there was only a very slight atrophy of the papilla, and yet vision was much diminished and the visual fields were distinctly affected. In still another instance there was a marked functional disturbance without ophthalmoscopic change. A few other authors have described distinct changes in the papillæ without any functional disturbance. The visual symptoms generally appear long after the others have been devoloped, but may come on with extreme suddenness (Uhthoff and Buss).

Gowers and Marie have called attention to the fact that the visual disturbance of multiple sclerosis may be unilateral.

Ophthalmoscopic examination has revealed several peculiar changes. Complete atrophy of the optic nerves is very rare. Uhthoff found it in only 3 of 100 cases. The rarity of this is worthy of note, and is probably due to the frequent preservation of the axis cylinders, in spite of the destruction of the medullary sheath. Incomplete atrophy—discoloration of the entire papilla—the peripheral parts being more distinctly altered than the nasal half, occurs in about 50 per cent. of all cases. Atrophy of the temporal halves of the papillæ is almost pathognomic of multiple sclerosis. Charcot insisted that the papilla retained a yellowish color in contradistinction to the white appearance in the atrophy of tabes. Optic neuritis has been found in a number of cases (in three of Luebbers' cases). Partial temporary atrophy is, according to Uhthoff, a common symptom in toxic amblyopias (tobacco and alcohol); but in these the atrophy is always bilateral, whereas it is frequently unilateral in multiple sclerosis. This special resemblance to the intoxication amblyopias is of interest, if we remember that multiple sclerosis has been shown to be at times of toxic origin.

The anatomical examination of the eyes (Uhthoff, Luebbers, and others) has revealed conditions as variable as are the functional disturbances. It has been shown that there are widespread atrophic changes in the optic nerves, depending probably upon the proliferating process emanating from the connective tissue, and gradually involving the nerve fibres; the sheaths are often destroyed, but the axis cylinder is, as a rule, not affected; in other words, the process is interstitial in origin, the changes in the nerves being secondary. In consequence of this preservation of the axis-cylinders, the secondary degeneration does not ensue, and the nerve fibres preserve their function.

The sclerotic changes in the optic nerves are much the same as in the other parts of the central nervous system. The next question is, whether or not the changes in the connective tissue are due to the disease of the blood-vessels. It is probable that the process begins differently, according to the varying etiology of the cases. Uhthoff, Buss, Taylor and others have paid attention to the behaviour of the blood-vessels. Several of these writers have found an increase in the number of the finer blood-vessels, and a change in their walls, followed by a proliferating process in the surrounding tissue.

Nolda, who studied the relations of multiple sclerosis to infectious diseases, thinks that the sclerotic patches take their origin from the walls in the blood-vessels. Luebbers, in comparing the changes in multiple sclerosis with those found in the primary optic atrophy of tabes dorsalis, finds that in multiple sclerosis there is more of a focal degeneration than in tabes, and, above all, there is in tabes no such number of normal axis-cylinders as in multiple sclerosis. The optic atrophy of tabes is due to the primary degeneration of the optic nerve fibres, while in multiple sclerosis the atrophy is the result of an interstitial neuritis, and this in turn may be due to a disease in the blood-vessels. In multiple sclerosis, it may be added, the retinal elements are not affected.

Scanning speech has long been considered a characteristic symptom of multiple sclerosis; but it is not the only speech disturbance in this disease. Putting it broadly, almost every form of dysarthria is possible in multiple sclerosis, for the disturbance is due to lesions in the pons or medulla interfering with the speech mechanism. Leube found a disturbance of innovation of the vocal cords in one instance, and Sachs has seen a typical case of the disease in which the opposition of the vocal cords was so imperfect that the patient spoke in whispers. In other cases nasal speech has been noted, and in still others true bulbar speech has been reported. There is often an evident speech tremor not unlike that observed in the early stages of general paresis, and resembling the peculiarities of speech in some patients suffering from Friedreich's disease. Disturbances of articulation, if excessive, are apt to be associated with other symptoms pointing to an involvement of the various nerve nuclei in the pons and the medulla oblongata (cases of Joffroy, Leyden, Berlin, Fuerstnar, Redlich and others). If these symptoms predominate, they constitute the special bulbar form of multiple sclerosis. This type was carefully considered by Hallopeau.

An interesting example of this class of cases was recently reported with an autopsy by Schuster and Bielchowsky. In this instance nasal speech was associated with paresis of the left arm and the left leg, with dyspnœa, headaches and double vision. There was also divergent strabismus of the right eye, as well as paralysis of both 6th nerves. The pupils and fundi were normal. A focal lesion in the pons was suspected, but the autopsy revealed multiple sclerotic foci in the pons and medulla and in other parts of the brain.

Aside from the peculiarities in the development of single symptoms, abnormalities occur in the development of the entire clinical picture of multiple sclerosis. In the literature of the subject the *formes frustes*, or aborted types, of multiple sclerosis, have given rise to considerable discussion. It is well to bear in mind that there is no regularity in the development of sclerotic patches, whence it follows that departures from the typical forms of the disease are frequent. Slightly irregular forms do not, therefore, deserve the designation *formes frustes*.

Charcot was the first to dilate upon these special types, referring particularly, (1) to cases in which certain symptoms, which had existed, had disappeared; (2) to cases of which the symptomatology had never been complete—such cardinal symptoms as the tremor or the nystagmus never having developed; (3) to cases in which the type has been altered by the intervention of other symptoms of a hemiplegic, tabic or amyotrophic order.

The establishment of the *formes frustes* has opened the door to diagnostic errors; for the multiple sclerosis may appear under the guise of an hemiplegia, of a transverse myelitis, or of an amyotrophic lateral sclerosis. In Sachs' opinion the diagnosis of multiple sclerosis should be made only if the cardinal symptoms of the disease, such as nystagmus, scanning speech, intention tremor are present in addition to the other symptoms of myelitis, hemiplegia, etc. The variability of the symptoms (Oppenheim) and the recurrent apoplectic and vertiginous seizures argue in favour of multiple sclerosis. Oppenheim has laid stress upon the occurrence in such cases of the characteristic changes in the fundus, with little or no functional impairment of vision.

Apoplectic attacks may occur in the course of a multiple sclerosis, but occasionally hemiplegia is developed slowly at the outset of the disease, and is then scarcely to be differentiated from other forms of apoplexy ; excepting that in these cases which affect men and women in middle life the usual etiological factors of an apoplectic seizure, cardiac disease, or specific endarteritis are generally absent. If the person who has suffered from an apoplectic attack develops marked nystagmus, scanning speech, or ataxic movement in the paralysis of the upper extremity, we may suspect multiple sclerosis. Cases of this character have been described by Werner, Bikeles and others. The question arises whether these apoplectic attacks in multiple sclerosis are due to vascular disturbances or to a peculiar localisation of the sclerotic patches. Sachs (with Charcot, Redlich and Babinski) believes that these forms are due to the early development of sclerotic patches in the pons and medulla oblongata, although Taylor has shown that such patches also occur in the cortex and other parts of the brain. Witzel has published a case in which there was a crossed paralysis of the facial and abducens nerve of the opposite side.

Many writers have conceded the occurrence of acute and chronic cases of multiple sclerosis, presenting none but spinal symptoms (Bourneville, Guérard, Leyden and Oppenheim have recorded cases of acute, transverse myelitis which were eventually proven to be cases of multiple sclerosis). In the absence of the ordinary etiological factors (transverse myelitis trauma, syphilis, vertebral disease) we may suspect disseminated sclerosis, but the cardinal symptoms of the disease only can prove the suspicion.

The purely spinal and chronic forms may simulate a spastic spinal paralysis. For months, and even years, no other symptoms may be forthcoming. No doubt, some of the cases of primary lateral sclerosis were in reality early forms of multiple sclerosis. Cases of this description have been reported by Krueger, Ormerod, Lapinski, and Redlich. Sachs saw a very typical case of this character at the Policlinic. In all such cases the occurrence of the characteristic changes in the fundus will lend valuable support to the diagnosis of multiple sclerosis. Multiple sclerosis may easily be confounded with other diseases, above all with cerebral spinal syphilis (Oppenheim, Sachs and others). The remissions in the symptoms, the preponderance of the spasticity over the paralysis, the apoplectic seizures are common to both diseases. Greater stress should be laid upon the pupillary changes in syphilitic affections (absolute immobility, unilateral pupillary symptoms, and irregular contour of the pupils), and the presence in multiple sclerosis of nystagmus, intention tremor, and scanning speech, which are rare in syphilis of the brain and of the spinal cord.

There is usually no difficulty in differentiating between multiple sclerosis and paralysis agitans; but Schultze and Sachs have reported upon cases in which the symptoms of multiple sclerosis have been associated with those of paralysis agitans; in one young patient of the latter, who exhibits most of the symptoms of paralysis agitans, there are also a nystagmus and marked nasal speech. In another patient, whose condition was at first diagnosticated (by competent observers) as functional tremor, then as intention tremor of multiple sclerosis, later developments have shown the disease to be a typical paralysis agitans. In view of such experiences, we may maintain that the clinical distinction between multiple sclerosis and paralysis agitans is not as absolute as it has been supposed to be since the days of Charcot.

Hysteria may simulate multiple sclerosis. More often hysterical symptoms are present in addition to those of multiple sclerosis. The differentiation will depend largely upon the presence of such distinctly hysterical stigmata as are foreign to the pure type of multiple sclerosis. Westphal's two cases, in which the anatomical changes were wanting, although the patients exhibited all the symptoms of the disease, have led to much discussion. French writers have called them hysterical without much hesitation, and Strümpell speaks of a separate form of pseudo-sclerosis, bringing under this head what appears to us a rather heterogeneous number of cases. He has allied with this form a diffuse sclerosis of children; but the clinical types which he attempts to establish need careful examination before they can be adopted.

As to multiple sclerosis in early life, the differential diagnosis between it and other diseases of infancy presents so many difficulties, that much care should be exercised. In a recent article by Strümpell, in which the author challenges criticism on other points, he very properly insists that the diagnosis of multiple sclerosis in children is not certain, unless corroborated by *post-mortem* findings.

The first case of multiple sclerosis in a child appears to have been described by Schuele (1871). This was followed by reports of Dreschfeld, Bristowe, and Ten Cate Hoedemaker. Marie, in 1883, reported upon cases of his own, and collected 13 in all. In 1887 Unger had increased this list to 19. Six years later, Totzke, reporting two cases of his own, tabulated 39 in all. One case was reported in Sachs' work on the "Nervous Diseases of Children," which Stieglitz has, through a printer's error, called a doubtful case in the latest publication on this subject. Stieglitz cites three cases of his own, and tabulates 35, evidently excluding some which he considered doubtful. In addition to those diseases of the adult which enter into the discussion of the differential diagnosis, such as syphilis, transverse myelitis, hysteria, we must especially consider the infantile spastic palsies, hereditary spinal ataxy (Friedreich's disease), hereditary cerebellar ataxy, and hereditary spastic paralysis—the last has been omitted from Stieglitz's carefully prepared list.

As for the infantile cerebral palsies, the ordinary paraplegias (birth cases and cases of defective development) will generally be ruled out by the history of early onset, by the frequent occurrence of convulsions, and by defective development, which is more marked than in cases of multiple sclerosis.

The acute hemiplegias, with their characteristic symptoms, cannot give rise to any confusion. Doubts could be entertained, however, with respect to the cerebral diplegias. Nystagmus is common enough in these; intention tremor, or, rather, ataxic movements, are observed frequently, and disturbances of speech (bradylalia) are very common. It is very rare, on the other hand, to find any two of these symptoms in a case of diplegia. Furthermore, it should be remembered that a cerebral diplegia of an acute onset is a rare occurrence, and rarest of all in patients beyond the age of five to seven years. These cerebral diplegias present extreme contractures and post-paralytic disturbances of motion (athetosis and the like), which seldom occur in multiple sclerosis.

Of the large number of cases of cerebral diplegia which Sachs has seen, there has not in a single instance been reason to suspect the presence of multiple sclerosis. In making the diagnosis, the entire congeries of the symptoms should be considered, and not any one clinical sign. Stieglitz says, correctly, that the question is whether infantile cerebral palsy has been excluded in cases diagnosticated as multiple sclerosis.

From Friedreich's disease differentiation is generally easily made, although every now and then patients come under one's observation whose clinical symptoms may leave some doubt as to the presence of one or the other of these diseases. A resemblance may be due to the peculiarities of speech, to the awkward movements of the upper extremities in Friedreich's disease, which may simulate an intention tremor, and to the presence of nystagmus; but the condition of the reflexes in disseminated sclerosis, the optic atrophy and the absence of true ataxia of the lower extremities will help to establish the differential diagnosis. In Friedreich's disease the disturbances of speech are rarely of the typical scanning order.

Sachs had an adult patient in whom the symptoms pointed to Friedreich's ataxia, and yet there was a bare possibility of his having a bulbar form of multiple sclerosis. This patient was 39 years of age, his past history was good, without any evidence of preceding syphilis. At the age of 20 he was weak in the knees, and frequently made mis-steps. In 1887 he had sought medical advice because of difficulty of locomotion, noticed especially in climbing stairs. When examined in March, 1895, he complained chiefly, while walking, of weakness in the extremities and slight difficulty in speech. He exhibited an ataxic spastic gait and static ataxia. The pupils reacted to light and accommodation; knee-jerks absent; distinct ataxia of the right upper extremity; speech midway between slow and bulbar; slight atrophy of the optic nerves. The case was at first supposed to be one of locomotor ataxia, but the further development of the disease showed that it was either Friedreich's ataxia or a bulbar form of multiple sclerosis. The intention tremor, the optic atrophy, and the peculiarities of speech, would be in harmony with the diagnosis of multiple sclerosis; but the absence of the knee-jerks, ataxia of the upper extremities, which was not a typical intention tremor, and the normal behaviour of the pupillary reflex, were in keeping with Friedreich's ataxia, which appears, however, as a rule, early in life. The first symptoms in this patient's case appeared at least as early as the age of 20 years, and possibly earlier.

Still more difficult is the differentiate of multiple sclerosis from hereditary cerebellar ataxia (type, Nonne-Marie). This is well illustrated by Stieglitz's Case 2, in which the diagnosis of multiple sclerosis hangs by a hair, and the author himself does not appear to be firmly convinced of the diagnosis. If a previous history of ptosis, of a paresis of the internal rectus muscle and the Rhomberg symptom do not support the diagnosis of hereditary cerebellar ataxia, it is also certain that swaying of the body, with the eyes open and closed, is not very usual in multiple sclerosis. In a case like this, as well as in the one cited above by Sachs, the *post-mortem* examination alone will satisfactorily determine the nature of the disease. It is well to insist again upon the fact that the mode of onset, the time of life at which the disease is developed, as well as the history of some preceding illness, will often help us to differentiate between the two diseases.

Hereditary, spastic paralysis, as described by Newmark, Pelizaeus, and others, should be considered in this connection. There is a striking resemblance in the symptoms, but nystagmus, intention tremor, and scanning speech are not typical of the hereditary form of spastic paralysis. Only a few months ago a boy, 12 years of age, came under my observation, who, after a fright, had developed marked spastic paralysis of the lower extremities, with slight ataxic movements of the right upper extremity, and a peculiarity of speech, which was slow and deliberate, rather than scanning. There was no nystagmus, no pupillary abnormalities, no optic atrophy—in short, no symptoms that were at all characteristic of multiple sclerosis, except the speech. In this case the fact that the boy was absolutely well until the age of 12, that there was no history of any similar occurrence in any member of his family, and that the disease came on in rather abrupt fashion, argued largely in favour of multiple sclerosis; and yet, but for the slight ataxic movements of the right upper extremity and the peculiarities of speech, there was little to throw the balance in favour of disseminated sclerosis. In cases of this description time is needed for the development of further symptoms, which will enable one to establish a diagnosis with some degree of certainty.

Etiology.—The author protests against the supposition that the disease, occurring at widely different periods of life, should or could be due to one cause only. The study of the subject has led him to the belief that there are several and equally important causes which may give rise to multiple sclerosis. Charcot and his immediate followers recognised the importance of exposure to cold and wet, and we may well concede that occasionally it is the refrigeration process, and not a mere wetting, which may be the cause of the disease. The same school admitted also traumatism and emotional depression. Mendel, basing his views upon four cases of his own and a collection of 24 others taken from literature, is in favour of traumatism, of late years several authors have also contended for this origin of tabes. Mendel concedes that there must be an acquired or congenital predisposition to the disease.

As to the emotional origin the author speaks of a young girl of 18, who developed typical multiple sclerosis after the death of a parent; and of a boy of 12, who had been frightened by a fire occurring near his dwelling.

The earliest writers have recognised that disseminated sclerosis, in contradistinction to paralysis agitans, is a disease of early life, the majority of cases occurring before the age of 30, and some of them before the age of puberty. During the past ten years much has been made of the occurrence of the disease in children; but the majority of cases should be looked upon with some reserve, though the disease may possibly occur. Women were supposed to be subject to the disease more frequently than men. In 39 cases collected by Berlin, 26 were women and 13 men. Such differences may have been accidental, for later statistics have shown a very different relationship between the sexes. In Nolda's collection of 36 cases, occurring in early life, 16 were boys and 10 girls. In Krafft-Ebing's collection there were 58 males and 42 females. In Redlich's experience the number was almost equally divided between men and women—12 males and 11 females. In Stieglitz's statistics there were 17 males and 17 females, the sex of one case not being mentioned. In the 15 cases seen by the author the ordinary division has been entirely reversed, for of these, 10 were males and only 5 females. Such smaller statistics are subject, however, to accidental variations, and it would not be fair to infer from them that the disease shows any distinct preference for sex.

Of late years the occurrence of multiple sclerosis after infectious diseases in early life has led to the theory of its infectious origin. As long ago as 1878, Leyden insisted upon the importance of preceding infectious diseases. Other authors referred to its occurrence after typhoid fever (Ebstein, Kahler, Pick, Westphal and others); after cholera (Joffroy); after smallpox (Charcot); but Marie has been the warmest advocate of the theory of the infectious origin of multiple sclerosis, and he contended more particularly that the primary change was to be looked for in disease of the blood-vessels. According to Redlich, who has gone over this part of the subject very thoroughly, up to the year of 1897 cases in support of this view were cited by Schoenfeld (after diphtheria), by Massolongo (after influenza), by Rolland (after phlegmonous angina), by Williamson (after rheumatism), by Torti and Angelini (after malaria). Redlich cites a case of polyneuritis of a boy of 17, coming on after a sore throat. The clinical symptoms in this case gradually passed into those of multiple sclerosis. Henschen refers to a case of a girl of 14, who developed the symptoms of multiple neuritis after diphtheria, death ensuing after three months. At the autopsy he found multiple sclerosis and degeneration in the peripheral nerves. This case needs careful interpretation, for it is, at least, questionable whether or not multiple sclerosis existed for some time before the diphtheria. The rapid development and early death make the case an entirely exceptional one.[1]

Among more recent authors, Oppenheim, Leyden, Goldscheider, and Sachs have favoured the occasional infectious origin of the disease, but do not feel warranted in supposing this etiological factor to be far more important than many others. Marie's views have been opposed more particularly by Redlich and Krafft-Ebing. In the 15 cases observed by Sachs only two came on after infectious diseases, and in both of these so long after such a disease that a direct relation, as of cause and effect, could not be established. It is well to bear in mind, however, that if infectious diseases were the chief cause of multiple sclerosis, the vast majority of cases should come on before the age of 15, and such is not the case.

Oppenheim has directed attention to the large number of cases of multiple sclerosis in which chronic metallic poisoning seemed to play a rather important part, basing his views upon the frequent occurrence of multiple sclerosis in those who work in factories in which toxic chemical substances are used—such as lead, tin, aniline dyes, and the like. It was Oppenheim, too, who referred to the importance of a puerperal condition as a predisposing cause.

It may be asserted with some degree of assurance that syphilis is not an important etiological factor, although Jacobson, Moncorvo, and Filatow have cited cases in which hereditary syphilis was present, and some others (Michaelow and Schuster) have referred to acquired syphilis; but Redlich and Sachs have noted that multiple sclerosis and multiple cerebro-spinal syphilis have so many symptoms in common that the differential diagnosis is not easily established. Whether or not the disease be distinctly hereditary is still in doubt, although Eichhorst has published two cases in which the disease occurred in mother and child. Its occurrence as a family disease has been noted by Friedreich and Erb (according to Krafft-Ebing and Redlich), and Totzke has mentioned the occurrence of the disease in two members of the same family. But the question naturally arises, since these cases were not substantiated, whether they may not have been cases of hereditary spastic paralysis.

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[1] Jurgens (*Berliner kl. Wochenschrift*, April 1, 1898) reports upon the case of a child, six months old, that had had convulsions at the age of three months; repeated convulsions with paralysis of right arm. Child died in convulsions. The brain contained "sclerotic patches," in which protozoa were found similar to those found in patches in the heart. The finding is of interest, but it has no bearing upon multiple sclerosis.

The latest contribution to this subject has been made by Blumenreich and Jacoby, who report on 29 cases of multiple sclerosis—23 in males and 6 in females—and in these cases the infectious diseases, intoxications, and traumatisms have been the chief etiological factors. They are of the opinion that the original cause may have preceded the outbreak of the disease for many years, and that the disease may even be of congenital origin. It is somewhat surprising to find that alcoholic and sexual excesses are not cited as occasional etiological factors of some importance; for, in the present writer's experience, two cases seemed to be the result of previous alcoholic excess, and in three now under observation no other factor except prolonged sexual excesses can be found. In these three cases, moreover, the clinical symptoms are so typical that there can be no doubt of the existence of multiple sclerosis.

A little reflection regarding the etiology of multiple sclerosis necessarily leads to the conclusion that many different causes may be at work resulting in the development of the disease, provided there is a predisposition to the disease. Sachs does not agree with Redlich in refuting Strümpell's theory of the endogenous character of multiple sclerosis, and thinks that for a large number of cases, particularly for those occurring early in life, some slight defect in the original development of the central nervous system must be held responsible for the outbreak of the disease, and that the other etiological factors so frequently referred to are to be regarded as the exciting causes. If we adopt this view, the usual resemblance between the cases of multiple sclerosis developing early in life, and the various forms of hereditary and family affections observed at that period, can be interpreted more readily.

Sachs also had occasion to observe several cases in which a distinct neurasthenic condition, due to overwork, preceded the development of the symptoms of multiple sclerosis. According to Edinger's theory,[1] the development of a sclerosis following upon functional exhaustion does not appear to be at all anomalous. In a patient, a music teacher, a functional tremor first appeared in the right hand; this was followed by a distinct intention tremor, and from this stage the symptoms passed into those of a typical paralysis agitans. Altogether, the relation of functional to organic nervous diseases would appear to need further attention, for the same sequence of events has no doubt been observed by others, and it is not to be supposed that the diagnosis of a preceding functional condition was erroneously made. Some of the mooted points regarding the etiology of multiple sclerosis acquire additional interest, if we consider them in connection with a discussion of the morbid anatomy and pathogenesis of the disease.

Pathological Anatomy and Pathogenesis.—The irregular sclerotic patches (plaques) occurring in different parts of the central nervous system constitute the chief anatomical feature of the disease. These "plaques" represent the terminal condition of a morbid process often lasting through a period of years; but a study of "late stages" has helped us a little toward an understanding of the morbid anatomy of multiple sclerosis, as has been the case in regard to poliomelitis. The process can be understood only if studied in its earliest stages. Such studies have been made by Ribbert, Cramer, Bikeles, Taylor, and Goldscheider.

With some slight exception, the general agreement has been reached that the blood-vessels play an important part in the initial stages of the disease. Goldscheider describes the conditions as presented by a patch in the cervical portion of the spinal cord. The blood-vessels are dilated, and the adventitia is infiltrated with small cells and with considerable detritus. In the vicinity of these blood-vessels the nerve fibres are swollen, the sheath and the axis-cylinder participating in the swelling. The medullary sheath suffers more than the axis-cylinder; at least, every cross-section exhibits a number of naked and well-preserved axis-cylinders. Goldscheider supposes that the enlarged nerve fibres crowd against each other, causing the disintegration and absorption of the myelin of some fibres, thus leaving room for other fibres to persist in their enlarged (swollen) condition. The changes thus far described are not unlike those occurring in an ordinary acute myelitis.

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[1] This theory appears to have received experimental proof; *cf.* Reports of the Congress f. innere Med. Wiesbaden, 1898.

Taylor is of the opinion that we are not warranted in connecting the vascular changes directly with the sclerotic process, for the patches are not always related to the diseased vessels, and in many plaques the blood-vessels are entirely normal, and other portions of the vascular system show no tendency to disease.

As early as 1863 Rindfleisch maintained the importance of disease of the blood-vessels. According to him, each plaque contained in its centre a blood-vessel with changed walls, denoting a condition of chronic inflammation. Borst found marked changes in almost every blood-vessel (hyaline degeneration, narrowing, thickening of the walls, etc.). He also found a condition of hyperlymphosis and lymphstasis, which he supposed caused a destruction of the medullary sheaths; but his views lack support. Redlich found changes in the larger blood vessels, and even in the capillaries; the neuroglia tissue is easily affected in the immediate vicinity of the blood-vessels, leading at times to a perivascular sclerosis. Ribbert found in two blood-vessels thrombi consisting of leucocytes. A plugging of blood-vessels was also noticed by Redlich; but these findings only prove that the vascular system is diseased in some instances. Other reports show that the blood-vessels often remain entirely normal. The inference to be drawn is that, while the blood-vessel may be the starting-point of the morbid process in some cases it need not be so in all. The final results are the only ones that are entirely similar in all cases; but there is a difference of opinion as to the intermediate stages by which these are reached. Thus, in Goldscheider's case the nerve fibres were the structures particularly affected; while in Ribbert's case the neuroglia were chiefly affected, and there was no swelling of the nerve fibres.

If the nerve fibre becomes involved, it is no doubt true, as was recognised by the earliest investigators (Rindfleisch, Charcot, Leyden, Schultze) that the medullary sheath is easily destroyed, while the axis-cylinder remains normal. Popoff alone contends that the axis cylinder is also destroyed, but that it is regenerated. This *may* occur at rare intervals; it is much more rational to assume that the axis-cylinder is not destroyed, and the preservation of normal axis-cylinders in a mass, exhibiting any number of disintegrating medullary sheaths, argues in favour of this view. The preservation of the axis-cylinder is responsible for the lack of secondary degeneration in multiple, sclerosis. Buss has recorded an ascending degeneration of the columns of Goll, and of the direct cerebellar tract from the eighth cervical segment into the medulla oblongata. These observations need further corroboration.

It has been assumed by Redlich and others that the fine fibrils which Popoff took to be regenerated axis-cylinders are part of the proliferating neuroglia tissues. It is also significant that Weigert, whose opinion is of the highest value in this matter, insists that the proliferation of neuroglia tissue is more marked in multiple sclerosis than in any other disease. This statement militates against the view of Lapinsky, that the fine fibrils discovered in the plaques are connective tissue formations issuing from the medullary sheaths, and are not neuroglia fibres. Redlich, using Weigert's neuroglia stain, has found that the proliferating interstitial tissue consists of neuroglia fibres of varying sizes. Huber admits slight changes in the neuroglia, consisting chiefly of lacunæ, due to the loss of nerve fibres, and of the presence of detritus and granular cells; the process is of parenchymatous origin, the changes in the neuroglia and in the blood-vessels being secondary. Adamkiewicz supports the theory of the primary affection of nerve fibres.

If the neuroglia be affected, primarily or secondarily, its cells, as well as its fibres, undergo proliferation. Redlich maintains that the neuroglia changes may be exhibited in various ways: (1) The increase is largely due to the increase of neuroglia fibres, which gradually form a network of fibres, in which only slightly altered nerve fibres will be found. As these neuroglia fibres grow broader and broader, the network becomes denser. (2) In some patches the network is not so dense, the neuroglia cells and their processes being the most pronounced features, while the nerve fibres have dropped out. This appearance is entirely similar to the account given by Huber, to which we have referred above.

Fuerstner says that the neuroglia cells are developed from leucocytes, thus reconciling the opinions that the disease is a parenchymatous affection and that it is of vascular origin. The actual participation of the neuroglia tissue has acquired greater interest, in view of Strümpell's recent theory that the disease is due to a primary proliferation of the neuroglia, that multiple sclerosis is practically a multiple gliosis, and that it belongs to the order of endogenous affections which are excited by accidental causes, such as injuries and infectious diseases. Ziegler appears to have entertained a similar view (Redlich).

To complete the record of historical changes, we may add that Gowers referred to the involvement of the ganglion cells in the earlier stages of the disease; while Taylor states very positively that the ganglion cells do not become affected until the late stages of the disease have been reached. The changes as described by Taylor in three cases of his own are: Considerable pigmentation, filling up the entire cell body, which is unusual in healthy young individuals; the nuclei are generally wanting; the nerve processes are well preserved; a diminution of cells. Koeppen found the ganglion cells normal. Obersteiner allowed that the ganglion cells may exhibit changes similar to those found in myelitis.

The patches of disseminated sclerosis present a bluish-gray appearance, and are sometimes slightly elevated; at other times the parts in which they occur appear shrunken and contracted. The question has arisen whether there is any rule governing the distribution of the plaques. By some it was supposed that the brain or the spinal cord may be affected singly. Charcot supported the view of a spinal form of multiple sclerosis. Erb accepted this on clinical grounds only. Charcot, Strümpell, Erb and many other writers considered that the white substance of the brain and spinal cord was the favoured site; that the gray matter was rarely involved. Charcot believed that the cortex of the brain and cerebullum is rarely affected. Taylor finds: (1) White and gray are affected without distinction. (2) There is no especially favoured site for the development of the sclerotic foci. (3) The cortex of the cerebrum and of the cerebullum is not exempt. The points (1) and (2) cannot be accepted unconditionally, as such statements must rest upon the examination of a larger number of cases. Charcot believed that the auditory, the olfactory and the optic nerves are the only cranial nerves affected; others (Cruveilhier, Skoda, Taylor) have found all, or almost all, involved. It is well known that the motor and sensory roots of the spinal nerves are often diseased, and Taylor has demonstrated the occurrence of a marked degeneration of the nerve fibres in the *cauda equina*.

The sclerotic patches *may* occur anywhere, but there can be little doubt that they *do* occur most frequently in the dorsal half of the pons, of the medulla oblongata (whence the occurrence of "bulbar forms"), in the white strands near the periphery of the cord and in the gray matter near the central canal. Obersteiner believes that the patches are lessened in number as we approach the lumbar portion of the cord. Leyden and Goldscheider describe a condition of diffuse sclerosis, or of a diffuse chronic myelitis, due to the confluence of numerous foci in the spinal cord. These authors also describe a purely spinal form of multiple sclerosis, which is the representative of the old-time chronic myelitis.

The sclerotic patches appear, on macroscopical examination, to be sharply differentiated from the surrounding healthy tissue, but, microscopically, the transition is a gradual one. Taylor has shown, however, that the division between healthy and diseased tissue may be strictly defined, and the same is true in Goldscheider's case.

The pathogenesis of multiple sclerosis is still obscure. In all probability there is no single mode of origin. Charcot believed that the disease was due to an interstitial inflammation, with a special proliferation of the neuroglia, the destruction of the parenchyma being secondary to this. Charcot, after Rindfleisch, ascribed some importance to the blood-vessels, and regarded them as a source of irritation. Marie published his views regarding the infectious origin of disseminated sclerosis, the exciting agents being carried through the blood-vessels. Ribbert thinks some noxious substances conveyed by the blood, set up circumscribed inflammatory lesions in different parts of the central nervous system. Mendel explained the traumatic etiology of some cases through the agency of blood-vessels. Fuerstner brought the blood-vessels into play by making them primarily responsible for nutritive disturbances in the nerve cells and fibres. Goldscheider says: "I believe I shall describe the nature of the morbid process most correctly by assuming the existence of a perivascular inflammation which injures the neighbouring nerve structures, causing more especially a disintegration of the medullary sheath, but allowing the axis-cylinder to escape. Later on, in the course of the disease, a reactive interstitial proliferation is established." He regards multiple sclerosis as a form of disseminated myelitis. Koeppen, Huber and Taylor do not attach so much importance to the blood-vessels, and Redlich is not convinced that the disease is always of inflammatory origin.

The vascular theory has been opposed by others. Adamkiewicz regards the disease as a parenchymatous degeneration. Weigert believes that the neuroglia changes are secondary to the affections of the parenchyma, and Strümpell, as we have seen, is inclined to regard the disease as being due, primarily, to a peculiar defective (?) development of the neuroglia, which is made evident in later years by especial exciting causes.

We must conclude that there is some truth in several of the views advanced. We believe that the vascular theory holds good, particularly for the cases developed after infectious diseases, after injuries, etc.; and, according to the intensity of the toxic agent, the nerve fibres or the neuroglia may be attacked first. This is in agreement with views expressed by Redlich. Congenital anomalies (Ziegler, Strümpell) may make the central nervous system an easier prey for the various toxic agencies and other injuries. But there still remains a considerable number of cases which would seem to depend rather upon functional exhaustion, and such exhaustion might affect primarily the nerve elements of the central nervous system. For such cases the assumption of a parenchymatous affection would be most plausible.

BIBLIOGRAPHY.[1]

ADAMKIEWICZ. "Die degenerativen Erkrankungen des Rückenmarkes." 1888.

AMESCHOT (Forme fruste). *Neurol. Centralbl.*, 1895, p. 732.

BASS. "Das Gesichtsfeld," &c. Stuttgart, 1896.

BABINSKI. "Etude anatomique," &c. Thèse de Paris, 1895.

BECHTEREW. "On Specific Disseminated Sclerosis," &c. *Archiv f. Psychiatrie*, vol. xxviii., 1896.

BERLIN. *Deutsches Archiv f. klin. Med.*, 1874.

BIKELES. *Obersteiner Arbeiten*, Vienna, 1895, part iii.

BLUMENREICH & JACOBY. *Deut. med. Woch.*, 1897, No. 28.

BORST. *Ziegler's Beiträge zur path. Anat.*, vol. xxi., 1897 (Hyperlymphosis, &c.).

BOULOGNE, P. (de Lillie). *Revue de Medicine*, 1896, p. 404.

BOURNEVILLE ET GUÉRARD. "Sclerosis en plaques disseminées," 1896.

BRISTOWE. *Med. Times and Gazette*, 1897.

BRUNS. *Neurolog. Centralbl.*, 1896, p. 520 (Relation to Myelitis).

BUZZARD. *British Med. Journal*, 1893.

BUSS. *Deutsches Archiv f. klin. Med.*, vol. xlv., 1889.

CHARCOT. *Gazette Médicale*, January 8, 1878. *Progrés Mèdical*, 1879. (Formes frustes).

CHARCOT. "Maladies du Système Nerveux," 1892, p. 399, vol. i.

CRITICAL DIGEST. BRAIN, 1896, p. 388.

DAMTSCH. *Neurolog. Centralbl.*, 1890.

DEJERINE. *Revue du Méd.*, 1884.

DRESCHFELD. *Med. Times and Gazette*, 1878, vol. i., p. 140.

EICHHORST. *Virchow's Archiv*, vol. xcvi., 1896. (Hereditary form.)

FROMANN. "Untersuchungen über die Gefässveränderungen," 1878. (Quoted by Redlich.)

FREUND. *Archiv f. Psychiatrie*, 1891, vol. xxii., p. 317.

FÜRSTNER. *Neurolog. Centralbl.*, 1895, p. 615. *Archiv f. Psychiatre*, vol. xxx., p. 1.

GOLDSCHEIDER. *Zeitschrift f. klin. Med.*, vol. xxx., p. 417.

GRASSET ET RAUZIER. Vol. i., p. 796.

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[1] For full reference lists consult articles by Redlich and Stieglitz.

GUNTHER NAGEL. *Münchener med. Wochenschr.*, 1897, No. 32.

GUTZLER. "Dissertation," 1895. *Neurol. Centralbl.*, 1896, p. 561. ("Traumatism as an Etiological Factor of Multiple Sclerosis.")

HACKNEY. *British Med. Journal*, March 6, 1897, p. 586.

HENSCHEN. *Revue Neurologique*, 1897, p. 195. *Centralbl. f. Nervenheilk. u. Psychiatrie*, 1897, p. 298.

HOEDERMAKER. *Deutsches Archiv f. klin. Med.*, 1897, vol. xxiii.

HUBER. *Virchow's Archiv*, 1895, vol. xcv.

JACOBSON. *Neurolog. Centralbl.*, 1895, p. 736 ("Relation of Syphilis to Disseminated Sclerosis").

KAHLER U. PICK. *Prager Vierteljahrsschrift*, 1897.

KAISER. Diss. Berlin, 1889. *Neurolog. Centralbl.*, 1889, p. 434 ("Trauma als ætiologisches Moment").

KÖNIG. *Berl. kl. Wochenschr.*, 1895, No. 39.

KÖPPEN. *Archiv f. Psychiatrie*, 1886, vol. xvii.

KRAFFT-EBING. *Wiener klin. Wochenschr.*, 1885, No. 51.

KRÖGER. *Neurolog. Centralbl.* 1889.

KUNN. "Ueber Augenmuskelstörungen bei der multiplen Sklerose." Hamburg-Leipzig, 1896.

LAPINSKY. *Zeitschr. f. klin. Med.*, vol. xxviii., 1895.

LEYDEN. *Klinik d. Rückenmarks-Krankheiten*, Berlin, 1875, vol. i.

LEYDEN-GOLDSCHEIDER. *Nothnagel's Path.*, vol. x., 1897, p. 448.

LUEBBERS. *Archiv f. Psychiatrie*, vol. xxix., p. 768.

MARIE. *Revue de Méd.*, 1883, p. 536.

MARIE. "Maladies de la Moelle," Paris, 1892, p. 108.

MASSALONGO, E. SYLVESTRI. *Revue Neurologique*, 1893.

MENDEL. *Deutsche med. Wochenschr.*, 1897, No. 7.

NOLDA. *Archiv f. Psychiatrie*, vol. xxiii., 1892 ("Relation to Infectious Diseases").

OBERSTEINER. "Nervöse Central-Organe, &c.," third edition, 1896.

OPPENHEIM. *Berl. kl. Wochenschr.*, 1888, 1891, 1896. *Neurolog. Centralbl.*, 1896, p. 43.

ORMEROD. *St. Barth. Hosp. Reports*, 1889, vol. xxiv.

POPOFF. *Neurolog. Centralbl.*, 1894, p. 321.

PRITCHARD. "Keating's Cyclopedia of Diseases of Children," vol. iv.

REDLICH. *Wiener klin. Rundschau*, 1895, No. 49-51.

REDLICH. *Centralbl. f. allgemeine Pathologie u. pathologische Anatomie*, vol. viii., 1897 (containing recent literature).

REDLICH. "Arbeiten aus Prof. Obersteiner's Laboratorium," 1896, No. 4 ("Zur Pathologie der multiplen Sklerose, &c.").

RENDU. *Gaz. Med. de Paris*, 1894, No. 52. *Neurolog. Centralbl.*, 1895, p. 734.

RIBBERT. *Virchow's Archiv*, vol. xc.

RINDFLEISCH. *Virchow's Archiv*, vol. xxvi., 1863.

ROSSOLIMO. *Deutsche Zeitschr. f. Nervenheilk.*, vol. xi., 1897.

SACHS, B. *N. Y. Medical Journal*, September 1, 1894; "Nervous Diseases of Children," 1895, p. 345; *Philadelphia Med. Journal*, February 5, 1898 ("The Relation of Multiple Sclerosis to Multiple Cerebral Syphilis and to Paralysis Agitans").

SCHÖNFELD. Diss. Berlin, 1888 ("After Diphtheria").

SCHULTZE. *Neur. Centr.*, 1884 ("Preservation of Axis Cylinders").

SCHULTZE. *Virchow's Archiv*, vol. lxviii., p. 120 ("Relation to Paralysis Agitans").

SCHWARTZ. "Die Bedeutung der Augenstörungen," &c., Berlin, 1898.

SCHUSTER AND BIELCHOWSKY. *Neurolog. Centralbl.*, December 1, 1897.

STIEGLITZ. *Am. Journal of Med. Sciences*, February, 1898 (contains literature of Multiple Sclerosis in Childhood).

STRÜMPELL. *Archiv f. Psychiatrie*, vol. x., No. 3.

STRÜMPELL. *Neurolog. Centralbl.*, November 1, 1896 ("Multiple Gliosis").

TAYLOR. *Zeitschrift f. Nervenheilk.*, vol. v., p. 1, 1894.

TOTZKE. "Multiple Sklerose im Kindesalter." Diss, Berlin, 1893.

TORTI AND ANGELINI. *Neurolog. Centralbl.*, 1893, No. 24.

UHTHOFF. *Archiv f. Psychiatrie*, vol. xxi., 1891 ("Study of Ocular Symptoms").

UNGER. "Ueber multiple Sklerose d. Central-Nervensystem im Kindesalters." Vienna, 1887.

WEIGERT. "Beiträge zur Kenntniss der normalen menschlichen Neuroglia." Frankfort on the Main, 1895.

WERDNIG. *Med. Jahrbücher*, 1888. *Neurolog. Centralbl.*, 1889, p. 432.

WESTPHAL. *Archiv f. Psychiatrie*, vol. ix., vol. xiv., 1883.

WILSON. *Br. Med. Journal*, Nov. 25, 1876.

WITZEL. *Revue Neurologique*, 1895, p. 313.

WILLIAMSON. *Med. Chronicle*, March, 1894 ("Vascular Origin").

BRAIN.

PART III., 1898.

Original Articles and Clinical Cases.

THE ACCURATE LOCALISATION OF INTRA-CRANIAL TUMOURS, EXCLUDING TUMOURS OF THE MOTOR CORTEX, MOTOR TRACT, PONS AND MEDULLA.

I.—CHARLES E. BEEVOR, M.D., F.R.C.P.

WHEN the Council of this Society honoured me by requesting me to open a discussion on the Localisation of Intra-cranial Tumours, it occurred to me that, as the subject was too vast to compress into the time at my disposal, and as I have had some cases under my care both at the National Hospital, Queen Square, and at the Great Northern Hospital, of intra-cranial tumours, the diagnosis of the localisation of which could not be described, speaking truthfully, as accurate, and as those tumours did not arise in the motor cortex, motor tract, pons and medulla, it would be of interest and would narrow the subject down by excluding these parts, especially as the localisation in them presents, as a rule, less difficulty.

In the remarks which I shall make, I have avoided collecting the various published views on the subject, and I shall bring before you only cases in which either a *post-mortem* has been made, or the position of the tumour has been verified by removal by operation.

Owing to the kind permission of the Physicians at the National Hospital I have been enabled to go through the *post-mortem* records for the last five years, and to utilise the clinical notes of the cases of tumour, and I have to thank my colleagues very much for their ready co-operation.

By the term "accurate localisation" I understand that the localisation is sufficiently exact to enable the surgeon to know whether the tumour can be exposed and reached, or whether it is too deeply situated to do more than a palliative operation.

FRONTAL.

Taking the frontal tumours first, they may be divided into (1) extra-medullary, and (2) intra-medullary.

Extra-medullary Frontal.

The three cases I have of extra-medullary tumour were all situated at the anterior end of the left frontal lobe. In one of the cases which was under my care at the Great Northern Hospital, the patient had right-sided fits, beginning with closure of the right eyelids, followed by turning of the head and eyes to the right.

In the other two cases, in one the *headache* was situated above the eye and behind the ear of the same side as the growth, while in the other the pain was occipital and radiated to the forehead, although in this case the growth grew from the frontal sinus.

Vomiting and *optic neuritis* or *atrophy* were present in both the cases.

The *mental condition* was normal in one case; while in the other memory and intelligence were said to be good, but later the memory failed and he became abusive and drowsy, but in this case the growth spread across to the opposite frontal lobe.

In the first case the sense of *smell* was normal, while it was abolished on both sides in the second.

In the first case there was no *paralysis*, but the patient had a feeling of falling to the right. The knee-jerks were equal and well marked.

In the second there was slight weakness of the leg opposite to the side of the lesion, and another curious symptom occurred, viz. :—forcible flexion of the head on the thorax.

There was no sensory disturbance, except that in the first case shooting pains and slight anæsthesia occurred in the area supplied by the trigeminal nerve of the same side as the growth.

Nystagmus was absent in both cases.

Intra-medullary Frontal.

In the intra-medullary frontal tumours, which in all the cases occupied the whole of the central white matter, and of which I have notes of five examples :—

Headache was noted in all cases, and was in four of them in the frontal region, and in one on the same side as the growth, and in one on the opposite, but the pain was also referred to the vertex in three cases, and in two cases to the occipital region.

Vomiting was noted in three of the cases, and *optic neuritis* in all.

Mental failure.—In three of the cases memory was bad and the mental powers failing; in three of the cases incontinence of urine was noticed, of which the patients did not seem to be aware, though in one of these cases the intelligence is described as bright and the memory good. In two cases affecting the left hemisphere—superior frontal, and superior and mid-frontal regions—words were forgotten and objects wrongly named.

The sense of *smell* was only once noted to be dull on both sides, but not lost.

Nystagmus was absent, except in one case where it was obtained on lateral movements.

Hemiplegia.—In two cases there was weakness on the opposite side, probably due to pressure, with fits affecting that side in one case.

In one anomalous case there was weakness in the face and limbs on the same side as the growth, with increased deep reflexes on the same side.

In two cases, where the growth extended back to the anterior limb of the internal capsule, there was difficulty in moving the tongue in one case, and the tongue went to the opposite side when protruded in the other.

Unsteadiness of *gait* was noted in one case and in another a tendency to fall backwards and away from the side of the growth, when the eyes were closed.

In one case there was wasting of the muscles supplied by the trigeminal nerve of the same side as the growth and slight anæsthesia of its sensory area. There were therefore two cases in which the 5th nerve was involved on the same side as a frontal growth, and probably due to pressure.

The mental symptoms were most marked in a case in which there was a small growth about one inch in diameter in the white matter of each frontal lobe in addition to a smaller growth in the centrum ovale of one side. Here the mental symptoms much resembled those of general paralysis of the insane.

CEREBELLAR.

Cerebellar growths are also best described as (1) extra-medullary and (2) intra-medullary.

Extra-medullary Cerebellar.

The five extra-medullary growths were situated in the posterior fossa, and all these cases were in the anterior part of the fossa up against the petrous bone, except one case which grew in the cerebellar fossa below the lateral lobe of the cerebellum. In all of them there was headache, vomiting and double optic neuritis, except that the first two symptoms were absent in one case.

The position of the *headache* was important; in one of my cases with a tumour two inches long, growing from the middle peduncle of the cerebellum and extending forwards and compressing the left half of the pons and crus, the pain was described as in the frontal and temporal regions, chiefly the opposite side, with tenderness in these parts; pain and tenderness was at times felt in the whole opposite side of the head including the occipital region; and also in both occipital regions and down the neck.

In a case of mine with a tumour the size of a pigeon's egg, wedged in between the right lobe of the cerebellum, the pons and medulla, the pain was referred to the opposite frontal and parietal regions; while in another almost identical case there was no headache at all.

In my case with a tumour the size of half an orange in the right cerebellar fossa pressing on the right lateral lobe of the cerebellum, the pain was limited to the right occipital region and radiated forwards affecting the right posterior quadrant of the head, but never the frontal region; at other times it extended down the right side of the neck and chest, once to the waist but not down the arm.

The *Mental* condition in all the cases was normal except in one where the patient was described as depressed and emotional.

Smell was normal in all cases except one, where it had been lost for eight years on both sides.

Nystagmus was present in all the cases, and it occurred equally in one case in which there was slight hemiplegia on lateral movement to either side, but only towards the side of tumour; in one case it was rotatory.

Weakness in the face and limbs with increase of the knee-jerk was noted on the same side as the tumour in two cases, and in one of these the patient could not stand without help and fell towards the hemeplegic side, while in the other no staggering or falling to one side could be made out. In another case there was no hemiparesis, but the patient fell to the same side as the lesion and backwards.

All the above cases were in the anterior part of the fossa. Where the tumour was in the cerebellar fossa the patient fell to the opposite side.

There was falling towards the opposite side, and slight weakness of the limbs on the same side in a patient of Mr. Lunn's with a tumour in the posterior fossa against the petrous bone, whom I saw at the Marylebone Infirmary and on whom Mr. Ballance successfully operated. At present it is one of the points on which we want further observation, viz.:—the side to which the patient falls in cerebellar lesions.

In none of the above cases was there any actual rotation of the patient on his long axis either in the direction of screwing in or screwing out. (For the sake of clearness I would recommend that cases of rotation should be described in terms of a screw with the proviso that the head of the patient corresponds with the head of the screw, which, moreover, is considered to be a right-handed screw; therefore the rotation in waltzing would be in the direction of screwing-in.)

Giddiness, or a feeling of movement was experienced in all the cases, except one, and sometimes objects seemed to move the same way as the patient, but the direction of the patient and of the surrounding objects was not constant enough to be of any use as a means of diagnosis.

The condition of the *knee-jerks* was variable: in those cases where slight weakness existed on one side, the knee-jerk was increased on that side, *i.e.*, on the same side as the tumour; but in two cases in which there was no hemiplegia the knee-jerks were variable, being usually both increased and if anything more on the side of tumour.

A symptom which is very important in the diagnosis of tumour of the posterior fossa is the implication of the *cranial nerves*, and the two which are most commonly involved are the seventh and eighth. In three of these cases, where the tumour grew from the middle peduncle or was situated between the cerebellum, medulla and pons, one or both of these nerves were involved. The flocculus is situated at this point and in some cases the growth has originated in this part of the cerebellum. In one case deafness and paresis of the same side were the only localising symptoms of a tumour growing in the anterior part of the posterior fossa.

The deafness in these cases is perosseal and a tuning-fork on the mastoid is not heard on the affected side.

In one of these cases the face was completely paralysed with electrical reaction of degeneration, while in the other two movements of the face are described as weak.

When the tumour occupies the part known as the cerebellar fossa there may be no facial paralysis and no deafness.

Of the cranial nerves the 5th and 6th nerves were affected in one case, causing slight anæsthesia of the forehead and paralysis of the rectus externus on the same side as the tumour; but in this case there was also defective swallowing and protrusion of the tongue to the affected side.

This involvement, or not, of the 7th and 8th on the same side will assist in the diagnosis of a tumour occupying the anterior part of the posterior fossa, from that of the inferior part—the cerebellar fossa.

The anterior wall of the posterior fossa is formed by the petrous part of the temporal bone and is separated from the cerebellar fossa by the descending part of the lateral sinus, and a tumour growing from the middle peduncle would press on the petrous portion of the temporal bone and on the 7th and 8th nerves; while a tumour in the cerebellar fossa would compress the lateral lobe but not these nerves.

Intra-medullary Cerebellar.

Of intra-cerebellar growths in one lateral lobe there are two cases, in one of which, during an operation by Mr. Horsley, a cyst occupying the right lateral lobe burst, and an infiltrating soft growth was felt, occupying the anterior and middle portions of the right lateral lobe; as the case is doing well—two years after the operation—the growth has evidently not progressed.

The other case was that of a growth, about 1½ inches in diameter, within the lateral lobe and destroying the corpus dentatum.

Of these two intra-medullary cases the *headache* was in the opposite frontal region in the first; while in the other it was in the back of the head and spreading down the neck and to the forehead, and with the pain there were attacks of laboured respiration, profuse sweating, and retraction of the head. *Nystagmus* occurred in the first case in looking towards the side of growth, and there was weakness of the lateral movements of the eyes to that side and internal strabismus; in the other case nystagmoid movements occurred in all directions.

In the first case there was *weakness* of the arm and leg on the side of tumour, with staggering to that side and backwards. The *knee-jerk*, on admission, was diminished, and was less on the side of tumour; while after the operation it was increased,—more so on the side of tumour—and at the same time the nystagmus disappeared. In the other case there was no paralysis, the knee-jerks were equal, and though there was reeling, and inability to stand with the eyes shut, it was not in any definite direction.

In harmony with the first case are the symptoms of the case reported by Dr. Acland and Mr. Ballance in *St. Thomas's Hospital Reports,* 1894, of an abscess in one lateral cerebellar lobe; where the eyes deviated away from the side of tumour, and there was nystagmus and diplopia on looking towards the same side, while the arm and leg were weak on that side with increase of the knee-jerk.

MEDIAN CEREBELLAR.

With regard to the diagnosis of tumours of the median cerebellar lobe, involving especially the superior vermiform process, it is difficult to separate their symptoms from that of pressure on the superior cerebellar peduncle which is often involved, as well as of pressure on the corpora quadrigemina. In one of my cases on which Mr. Ballance operated, where the growth grew from the lateral lobe and compressed the median lobe and the superior peduncle, the headache was frontal, nystagmus was on lateral movement; there was no weakness of either side, but both erector spinæ were weak; the knee-jerk was absent on the side opposite to the growth, and the patient fell, in trying to stand, to the opposite side; but the most curious symptom was that of rhythmical movements of flexing the head on the trunk, opening and closing of the eyes, and flexion and extension of the trunk.

CORPORA QUADRIGEMINA.

In cases where the corpora quadrigemina are the seat of tumours, or are compressed by a growth from the median lobe of the cerebellum, the first symptoms are some paralysis of the ocular muscles, followed by staggering and falling forwards and to the opposite side, as in a case I published in

BRAIN in 1881, but as in this case there was compression of the right crus cerebri and the right superior cerebellar peduncle, it was difficult to apportion the cause of the staggering. Two other symptoms which are observed in tumours of the corpora quadrigemina are marked incoordination, together with loss of the sense of the position of the limbs, upper and lower.

These symptoms in association with ocular paralysis are strongly in favour of tumours of the corpora quadrigemina.

Many of these cases are mentally very slow, but this may be due to the hydrocephalus, which is sometimes present from pressure on the iter.

LATERAL VENTRICLES.

The question of tonic attacks of *opisthotonos* as a symptom of cerebellar disease is one of much importance, and in the cases which I have gone through it has not occurred so often as one might expect. It occurred slightly in an intra-medullary lateral lobe tumour as a drawing back of the head, and in one of my cases, where the growth, beginning in the middle cerebellar lobe on the upper surface, extended forwards, compressing the corpora quadrigemina and the right crus cerebri and right optic thalamus. I have, however, met with this symptom in two cases of distension of the ventricles, in one of chronic hydrocephalus occurring in the adult, and in the other, a case of hydatid of the right lateral ventricle in the posterior cornu, both of them at the Great Northern Hospital.

In the case of hydatid of the right lateral ventricle, the patient was a boy age 16. The onset began gradually six months before admission with headache, vomiting, and double optic neuritis + 4D., and later with deficient vision, attacks of giddiness, with a feeling of going round and falling backwards, followed by fits of unconsciousness lasting a few minutes. The chief symptoms were staggering and falling, as a rule, to the right, the same side as the lesion, but without any paralysis. Ankle clonus was, however, present, and more on the right than the left, though the knee-jerks were both increased and equal. The headache was over the right eye, in the right temple or in right forehead, but no tenderness. There was conjugate nystagmus on lateral movement, and especially towards the side of lesion. Convergence was not possible, the left eye diverging. In walking, he staggered, if anything, towards the side of lesion, though he also fell the opposite way at other times. He had several attacks of fainting and of giddiness; and in three he was unconscious, passed his water, became rigid all over, and had arching of the back from tonic spasm of the erectores spinæ. Mentally, he was dazed, confused and very lethargic, passing his water in bed. On three occasions there was marked inco-ordination of the left hand, and once tremor; but no paralysis of the limbs, though the left side of the face did not move quite so well as the right. Though the convolutions on the outer surface of the occipital lobe were thinned down to paper, no hemianopia was ever observed.

The other case of internal hydrocephalus, occurring in a boy age 16, gave symptoms very much resembling cerebellar disease.

The duration was twelve months, with gradual onset of headache, frontal and occipital on both sides, vomiting and optic neuritis, equal in the two eyes. In walking, he fell to the right. He had several attacks of vertigo, with rotation of objects from left to right. Attacks of headache occurred, with tonic spasm of the erector spinæ and arching of the back, and on examination the right erector spinæ was felt and seen to contract more than the left, with slowing of the pulse—on one occasion as low as 40—while the face was flushed.

On *post-mortem*, both lateral ventricles were evenly distended with fluid, and the ependyma was thickened and inflamed.

These two cases show that staggering to one side, and tonic spasm of both erectores spinæ may be caused by distension of one lateral ventricle, and that the unilateral symptoms of falling to one side and spasm of one erector spinæ may be caused by a bilateral distension of the lateral ventricles. Is it possible that these symptoms can be referred to pressure on the cerebellum by the distended ventricle?

With regard to cerebellar and frontal growths there are two parts of the brain, tumours of which are very difficult to diagnose, these are tumours of the optic thalamus and of the corpus callosum.

Optic Thalamus.

In one case of an abscess of the optic thalamus, there was severe pain in the occipital region of the same side, vomiting, double optic neuritis, with some weakness of the face and limbs of the opposite side, but the symptom which allied this case to a cerebellar case was the tendency of the patient to rotate to the side of the lesion (the right, or in the direction of screwing-in). At the same time there was pleurosthotonus and a tendency to fall backwards and to that side; while the symptom, which was associated with that of frontal tumours, was the slow mental condition.

Corpus Callosum.

Tumours of the corpus callosum always present great difficulty in that they are attended by marked mental symptoms. A valuable paper by the late Dr. Bristowe, in Brain, part xxvii., gives the following symptoms:—Headache, hemiplegia—first on one side then on the other, dementia with drowsiness and stupidity, optic neuritis and vomiting being often absent. In two cases at Queen Square there was headache at the vertex and on one side, optic neuritis in both cases, vomiting in one case, but not severe; mentally, one was very dull with delusions of suspicion, and the other had a vacant look; with regard to the hemiplegia, it seems to differ according to whether the anterior part of the body, or the posterior part, of the corpus callosum is affected. In one case where the posterior part was affected and the growth passed into both optic thalami without involving the internal capsules, there were fits, probably epileptic, no paralysis, but some slight hemianæsthesia; while in another affecting the greater part of the corpus callosum there was dragging of one leg, and six months later there was a partial paralysis of both orbiculares oculi, and both upper extremities, with rigidity of both legs; so that in this case there was a double hemiplegia.

OCCIPITAL, TEMPORO-SPHENOIDAL, SUPRA-MARGINAL, AND ANGULAR.

With regard to the occipital lobe, I regret that I have not a case for illustration, the notes of one very good case at the National Hospital having been lost at the time of the death of Dr. James Anderson.

Of temporo-sphenoidal lobe tumours there was a case in which the symptoms were headache, forgetting names of places, using wrong words, and a curious loathing of colours except green, with deafness of the opposite ear and hallucinations of hearing.

The growth was in the left temporo-sphenoidal lobe, but extended subsequently and produced other symptoms.

In tumours of the supra-marginal and angular gyri, the symptoms are word-blindness, and if the lesion extends deep enough to involve the optic radiations, hemianopia, as in a case of mine of abscess operated on by Mr. Horsley *(Ophth. Soc. Trans.)*, and as in the case of Dr. Colman and Mr. Ballance *(Clin. Soc. Trans.)*.

General Diagnosis.

The greatest difficulty occurs in the diagnosis of tumours in the frontal and cerebellar regions, as well as in the optic thalamus, in the corpus callosum and in the lateral ventricles.

Tumours may occur without giving rise to the symptoms which would be expected, and tumours may give rise to symptoms which are associated with other parts of the brain, and tumours may occur without giving any symptoms at all, although situated in parts of the brain whose functions are considered to be known. The difficulty at present in some cases is that after the position of the tumour is ascertained by *post-mortem*, one feels that given the case over again with the localisation known, the symptoms presented are not always those we are led to expect from our present knowledge, and it would not be possible to diagnose the case.

The first point is the localisation of the part of the brain involved, and the second, and often more difficult point, the side of the brain which is attacked; and given headache, vomiting and optic neuritis, what are the symptoms which would enable one to make a diagnosis of the part involved? In the absence of any external signs, as softening or bulging of the bones, the diagnosis is often very difficult, and local tenderness to pressure or percussion is often deceptive, unless it is very constant.

Taking frontal tumours, I would say in summing up, that the headache in most of the cases is frontal, and if anything, is more on the side of the tumour than on the other, but that the pain is also vertical and occipital; that mental changes are usually well marked in intra-medullary cases, but not in extra-medullary, and is most marked when both frontal lobes are involved; that sense of smell is not lost in intra-medullary, but may be in extra-medullary cases, especially if the growth involves the under surface of the frontal lobe; that nystagmus, staggering to one side and hemiplegia are absent in extra-medullary, but may be present in intra-medullary, and though the last may indicate the side of the tumour, it cannot be relied upon. Attacks of giddiness are not common. With regard to the mental condition, the frequency of involuntary micturition is remarked, and is due to the mental condition rather than to any affection of the sphincters.

The question as to which frontal lobe is affected is often very difficult, but is assisted by the sense of smell being lost on one side only; by aphasia, which may be of the sensory and is not always the motor form; by one-sided fits, though these are usually absent; by implication of the 5th nerve on the same side as the tumour.

It must be remembered that in some cases the gait is unsteady, and if there is hemiplegia the patient may fall to that side.

In cerebellar growths of the lateral lobe the extra-medullary form which occupies the cerebellar fossa, presents pain and tenderness in the occipital region on the same side, the sense of smell and the mental condition are normal, or the latter is affected late in the course, nystagmus is present on lateral movements, there is no hemiparesis, staggering occurs backwards and to the opposite side, and giddiness is frequently present. There is no paralysis of the facial and auditory nerves.

In extra-medullary tumours occupying the anterior or petrosal part of the posterior fossa, the headache may be frontal, on the side opposite to the tumour, hemiparesis on the same side with increased knee-jerks, staggering either to same side or opposite side, with nystagmus lateral to the same side. Facial paralysis and perosseal deafness occurring on the same side, with lateral nystagmus to that side, and hemiparesis of the same side with staggering to one side, especially the same, are strong evidence of a growth in the petrous portion of the posterior fossa on that side.

Paralysis of facial and auditory nerves is of more localising importance than a doubtful staggering.

In intra-medullary tumours of the lateral cerebellar lobe, the pain is occipital, or in the opposite frontal region; there is hemiparesis and increased knee-jerk on the same side with the conjugate movements to that side weak, and nystagmus on trying to look to the tumour side.

Drawing back of the head occurs in cerebellar cases both intra- and extra-medullary, but I have not seen opisthotonos, though it is present sometimes.

In tumours of the optic thalamus, the pain is occipital on the same side, the mental powers are slow, weakness and nystagmus of ocular movements to the same side, weakness of opposite limbs, rigidity of limbs of same side, staggering backwards and to same side, and attacks of the rotation of the trunk with the face towards the same side.

In tumours of the corpus callosum the headache is vertical, there are mental symptoms, chiefly stupidity and slowness coming on early, followed by double hemiplegia, with one side affected before the other.

In distension of one lateral ventricle at its posterior part, the symptoms are:—Frontal headache on the same side, mental condition stupid and lethargic; nystagmus on lateral movement, more towards same side; staggering to both sides but rather more to same side; ankle clonus on both sides but especially on same side; inco-ordination of opposite arm; opisthotonos with spasm of erector spinæ of both sides.

The following are some of the points on which it is hoped that the discussion of this paper will throw some light:—

The relation of the position of the headache to:—(1) extra-medullary tumours; (2) intra-medullary tumours.

The direction of staggering in relation to:—(1) cerebellar tumours of the cerebellar fossa; (2) cerebellar tumours of the petrous part of the posterior fossa; (3) cerebellar tumours of the median lobe.

Hemiparesis and its relation to the side of the cerebellar growth.

I have to thank Dr. Colman and Dr. Risien Russell for their abstracts of the notes of the cases in the autopsy book at Queen Square Hospital, and to Dr. Whiting and Mr. Goring for assistance in the notes of the cases at Queen Square and the Great Northern Hospital.

II.—J. Michell Clarke, M.D., F.R.C.P.

In bringing forward the following cases of intra-cranial tumour which have come under my observation, I have selected those only in which a *post-mortem* has been made, with one exception in which the position of the tumour was confirmed by operation. Dealing first with the diagnosis of tumours of the præfrontal area of the brain, in these cases marked mental changes are often observed, and it has been thought that these mental symptoms are sufficiently characteristic to warrant the localisation of a tumour in this region. Dr. Williamson, in his analysis of fifty cases,[1] found that mental symptoms were generally well marked, and in many cases they were the earliest and most pronounced symptoms. As the frontal lobes form one of the so-called "silent" areas of the brain, tumours of which may give rise to no focal symptoms, although after a time focal symptoms are often produced by spread of the growth to neighbouring parts, mental symptoms, if characteristic, would afford valuable help in diagnosis. The main difficulty seems to lie in the common occurrence of mental changes in tumours of other regions of the brain, so that in any individual case it is hard to say whether the mental symptoms present are more than may accompany an intracranial tumour in other situations. The following five cases of tumours of the præfrontal area have come under my observation:

In the first three of these cases the cortex of the frontal lobe was chiefly involved; in two, the growth was a gumma; in one, it involved the second and third frontal convolutions in the anterior two-thirds of their extent, and was dense and fibrous in consistence with nodules of almost cartilaginous hardness, and in the other affected the cortex and subjacent white matter to the depth of nearly half an inch of the middle and anterior parts of the second and third frontal convolutions, together with, on the under surface of the lobe, a small portion of the adjacent orbital convolution. In the third case the exact nature of the growth was not determined at the operation, nor could its whole extent be made out, but it appeared to be a diffuse gliomatosis affecting chiefly the posterior ends of the first and second frontal, and the upper part of the ascending frontal convolutions.

Headache was intense in all three; in one case most marked in the frontal; in another, in the frontal and occipital regions on the side of the growth. There was tenderness on the same side in two cases, in the temple and frontal regions respectively.

Vomiting of cerebral type occurred in all.

Epileptiform fits occurred as an early symptom in all; in two, as the first symptom; and were general in character at first, but in the third case they became later of Jacksonian type, beginning in the leg or foot, and in another case they later affected the opposite hand and arm most.

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[1] Brain, 1896, p. 345.

Paralysis. Hemiparesis of the opposite side present in two, developing into complete hemiplegia in one of them (case iii.). Absent in the other.

Sensation unaffected in all.

Gait somewhat uncertain and ataxic in type in one,; in the others only modified by extent of hemiplegia present.

Knee-jerks normal in one; exaggerated on both sides, but most on the opposite, in two.

Pupils. The opposite pupil was larger than the other in each case. In one case there was spasmodic contraction of the opposite orbicularis palpebrarum. No other cranial nerve was affected.

Optic neuritis was present in all; intense in two, most marked on the side of the growth in these cases; failure of vision in these eyes was an early symptom.

Other special senses unaffected.

The pulse was slow in two cases.

Bladder and rectum. No loss of control occurred in either case until late on in the illness. One patient suffered from a passing attack of diabetes insipidus.

Mental symptoms. In all three mental symptoms occurred early, and took the form at first of failure of memory and of mental dulness or deterioration.

Case I.—The patient was at first dull and heavy, slow in answering questions, forgetful, and at times irritable and dissatisfied. Later he was very slow in all mental processes, and appeared incapable of sustained attention. As a rule he was quiet, but in the early morning was occasionally restless and excited, or even noisy and quarrelsome if opposed. He awoke from sleep confused, not knowing where he was, and on one occasion, in the middle of the night, went out into the street shouting. Apart from the general signs of an intra-cranial tumour, these mental changes formed the most prominent feature of the illness in this case, and there were no localising symptoms, unless the mental ones could be so considered.

Case II.—The patient lay in a somewhat dazed condition during the day and was with difficulty persuaded to eat. He answered sensibly though slowly to questions, but appeared to have some difficulty in recalling the names of objects. At night he was restless, constantly trying to get in and out of bed, and when he got out of bed was several times unable to find his way back to it. Later he was for a week or two restless and noisy, delirious at night, shouting to imaginary persons, and for a time passed into a condition of almost maniacal excitement, with constant incoherent talking. This condition gradually passed off, and the patient finally left the hospital practically well, except that he was blind, to die suddenly a month later whilst walking in the street, without having had any return of his symptoms.

Case III.—As a rule mental condition dull and drowsy, but sometimes the patient was very hilarious, laughing at whatever was said to him. There were some signs of motor aphasia in this case. Later he grew irritable and restless, sometimes swearing, using violent language; at others, being sullen and suspicious of poisoning. This passed into a maniacal condition in which he required two attendants to restrain him. These mental symptoms were relieved to a considerable extent by the operation undertaken with a view of removing the tumour; afterwards he remained mostly quiet, but was at times irritable.

In *Case IV.* a large round-celled sarcoma sprang from the white matter of the right frontal lobe, and bulged into the anterior horn of the right lateral ventricle; the whole of this ventricle was distended. This patient had suffered from epileptic fits, general in character, which had followed an injury to the head twelve years previously.

The symptoms produced by the tumour were of six months' duration, and were briefly: failure of memory, intense headache in the frontal and in the occipital regions, on the same side as the growth, with some tenderness over the occiput. There was paresis of the left arm and leg, but the face was not affected. Paresis of right external rectus muscle. Marked optic neuritis. Exaggerated knee-jerks on both sides. No affection of sensation. Loss of control over bladder for one month before death. The patient was admitted a fortnight before the fatal termination of the illness which ended in coma. He was childish, and his memory appeared to be very bad; dull and drowsy in the daytime, he was restless and delirious at night, constantly trying to get in and out of bed. According to the history obtained of the illness, early symptoms were, failure of memory and attacks of "strangeness" or "wandering in his mind."

The symptoms common to these cases were epileptiform fits, general in character, and occurring early in the history of the illness; more or less intense optic neuritis, in two most marked on the side of the tumour; marked signs of mental disorder as an early and persistent feature of the illness, with increasing somnolence in the later stages; headache and sometimes tenderness in the frontal or occipital region on the same side, and later opposite hemiparesis, in one amounting to hemiplegia. Dr. Williamson (*loc. cit.*) states that no conclusion could be drawn from the cases he analysed as to whether mental symptoms are the result of lesion of the cortex or of the white matter. He summarises these symptoms as "condition of mental decadence or dull mental state; loss of power of attention; loss of spontaneity; the patient taking no notice of his surroundings; sleeping during the greater part of the day or being semi-comatose." In my cases, further features were the occasional irritability of temper and the nocturnal restlessness or delirium. In one case I observed that hilarious mental state with unmeaning laughter, affording a remarkable contrast to the patient's miserable condition, which was described by Oppenheim, and which Bruns[1] mentions as a diagnostic feature of frontal tumours. I think, then, that if, with the general signs of an intra-cranial tumour, mental symptoms of the kind above described appear early in the case, persist more or less throughout the illness, so as to form a prominent feature of it, and there are also frontal or frontal and occipital headache, and later signs of hemiparesis with or without localised convulsions, there is good evidence of a tumour of the præfrontal area. An additional symptom would be ataxia of gait.

It may be impossible to determine which side of the brain is affected. The presence of aphasia, or of paresis, or convulsions of the opposite limbs would be a guide, and in the absence of these symptoms, two others seem to occur with some frequency in these cases, namely, tenderness over the frontal region on the affected side, and greater intensity of the optic neuritis on that side. The occurrence of occipital headache in frontal tumours is important, because there may be difficulty in the diagnosis between a frontal and cerebellar tumour, especially if, as is sometimes the case, there is ataxy and loss of the knee-jerk in a case of the former. Some diagnostic indications are given by Dr. Williamson (*loc. cit.*). But, as my first case shows, a tumour of the præfrontal area may give rise to remarkably few symptoms, and occasionally apparently to none at all, as in the case of a woman who died in the gynæcological ward of the Bristol General Hospital, of multiple sarcomatous tumours, the primary growth being in the uterus, and secondary growths in the right ovary, pelvis, lumbar glands, supra-renals, liver, and a large growth in the right frontal lobe. There were at any rate no conspicuous symptoms of an intra-cranial tumour during life, and the existence of such a tumour was never suspected. Examination of the optic discs after death showed that there had been no optic neuritis.

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[1] *Die Geschwülste des Nervensystems.*

The growth, a round-celled sarcoma, was seated at about the centre of the frontal lobe, under the middle frontal convolution chiefly; it was about the size of a plover's egg, with its long axis antero-posteriorly. On the external surface the growth could be seen at the bottom of a furrow separating the superior from the middle frontal convolutions. Internally it lay against the anterior part of the lateral ventricle which was pushed inwards and downwards, the anterior end of the corpus callosum was pushed upwards and a little inwards. The growth appeared to have gradually displaced the adjacent parts without actually destroying them or interrupting the course of the fibres.

With regard to the optic thalamus, I have had one case in which the tumour, a glioma, was well defined and entirely limited to the left optic thalamus, occupying chiefly the middle portion, but extending also, though to a less degree, into the anterior and posterior nuclei of this ganglion.[1] The patient was a child, aged 6. The illness began with gradually increasing weakness of right arm and leg. Briefly, the chief symptoms were: headache on left side; vomiting; slight double optic neuritis; early loss of control over urine and fæces; paresis and tremor of right arm and leg, most marked in the arm, the position of these limbs being that of ordinary hemiplegia; the right side of the face was slightly affected at a late stage of the illness; the arm muscles showed some rigidity; the tremor of the right arm was "intentional" in character, resembling that of disseminated sclerosis. There was no ataxy of movement; no affection of sensation nor of the muscular sense. The pupils were equal, and acted well; no oculo-motor palsy; the visual fields could not be taken. Pulse 54-60.

One week before death.—Intermittent attacks of spasm of muscles of right arm and of neck came on, unattended by loss of consciousness, in which the right forearm was flexed and pronated, the fingers extended and spread, and the head drawn downwards and to left. The arm showed vaso-motor disturbance, being cold for two days, then red, swollen, hot, burning and painful to touch or on movement. There was left ptosis, and dilated pupils, which no longer reacted to light. The patient lay on the left side with the head and eyes at first directed to the left, later to the right.

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[1] A short account of the case was published in the Hospital Reports of *The British Med. Jour.*, 1891.

According to Schiff's and Nothnagel's experiments on unilateral lesions of the optic thalamus, the head looks towards the healthy side, and on this side the flexors, whilst on the opposite (affected) side the extensors of the upper extremity are contracted. In my case the limbs on the side of the tumour were unaffected.

I have found in recent literature eight other cases in which the tumour was fairly limited to the optic thalamus, and another in which both thalami were involved, but the right chiefly. Including the case given above, this brings the number to ten. As regards paralysis, there was paresis of the opposite limbs in nine cases, affecting the arm and hand most in eight. In six movements of the opposite arm or hand were observed, in three of them as a well marked "intention" tremor; in one the movements were choreiform, and the arm "always on the work;" in another shaking of the arm is described; and in the fifth incoordination of movement; in this case the fingers were found in the position of athetosis, as if they had slowly moved there. In four of the cases the movements are distinctly stated to have been diminished by rest. Tonic spasm of the opposite forearm or arm was present in three cases.

Anæsthesia is only mentioned in two cases, and then was not complete. Incontinence of urine appeared early in four cases, and was absent, or absent until the end of the illness, in six. The knee-jerk was exaggerated in four cases, in two of these on both sides; in one there was ankle-clonus. Optic neuritis was present in six, in two most intense in the opposite eye, in one in the eye on the side of the tumour. Hemiopia is not mentioned in the reports of most of the cases. Mental slowness or dulness was noted in four cases, and in one there was mild acute mania. Convulsions occurred in one case. The condition of the pupils was variable. Ptosis was a late symptom in two cases, diplopia and nystagmus were each observed once.

The chief points in diagnosis in the above cases seem to be the infrequency of anæsthesia on the opposite side, which is rather contrary to expectation, and of early loss of control over the bladder (in four out of ten only); the constancy of hemiplegia on the side opposite to the tumour, or rather hemiparesis, for the paralysis does not seem to have been absolute in any case, except in the upper extremity, in which it was most pronounced, and the affection of the same arm with a tremor, of intentional or athetoid type. The very marked vaso-motor symptoms in the arm in my case are interesting to bear in mind.

In addition Bruns[1] lays stress as a symptom of importance on paralysis of the emotional movements on the side of the face opposite to the lesion. I believe that the tremor abovementioned is next most often observed in tumours of the cerebellum; other signs would probably be present in such a case which would aid the diagnosis.

Cerebellar tumours.—In three cases a well-defined tumour was limited to one lateral lobe of the cerebellum; in two of them the tumour grew from the inferior part of the lateral lobe; in one partly involving it, but being in its largest part outside the lobe, in the second occupying the posterior third of the lobe, and in the third case the centre of it.

Case I.—This tumour occurred in a boy, aged 15, who had been under my care for some years, with well-marked but stationary symptoms of Friedreich's disease. The signs connected with the growth of the cerebellar tumour were so well defined and distinct from those due to hereditary ataxia that I have ventured to use the case from the point of view of the cerebellar tumour. (I append a brief abstract of the cerebellar symptoms, the case having been previously reported in *Brit. Med. Journ.*, 1894.)

During July he suffered from intense headache affecting the right occipital and the frontal regions, not distinctly limited to either side in the latter, vomiting, and epileptiform convulsions. When admitted in August, eighteen days before his death, he could neither stand nor walk, and fell to the left when he attempted to do so. He was dull and heavy, and had incontinence of urine. He lay in bed curled up on his left side, with the lower extremities flexed at the hips and knees; he seemed unable to lie in any other position, and if placed on his back or right side, at once turned round on to his left side again, apparently quite involuntarily. This he would do again and again. The eyes were as a rule, but not constantly, directed to the left. Knee-jerks absent, they had been present, though feeble, until July. Paresis of right 6th nerve. Retraction of head and neck and rigidity of posterior neck muscles.

Four days before death he suffered from attacks, lasting a few minutes at a time, of marked opisthotonos, the body being supported only on the back of the head and the heels; these attacks increased in frequency, until for the last twenty-four hours of his life he lay constantly in a position of extreme opisthotonos. During these four days he was unconscious.

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[1] *Loc. cit.*

Post-mortem.—There was found springing from the under surface of the right lobe of the cerebellum a round, somewhat lobulated tumour, which lay beneath the cerebellum by the side of the pons and medulla. The neighbouring parts were pushed aside but not destroyed by the tumour, which was a round-celled sarcoma of soft consistence, and distinctly demarcated from the surrounding cerebellar tissues. On making vertical sections from before backwards the growth was found to be largest at the mid-point of the right lateral lobe of the cerebellum. The flocculus and lobules on the inferior surface of this lobe were destroyed, and its central white matter flattened, compressed, and partly destroyed. Microscopic sections of the pons showed that the posterior fibres of the middle peduncle, especially those lying uppermost in the pons, were fewer in number on the right than on the left side. There was no meningitis.

Case II.—This case was one of a fibroma, which *post-mortem* was found to project as an oval mass about 2 inches long and 1½ inches in diameter, from the inferior and posterior surface of the left lateral lobe of the cerebellum; it was well defined and demarcated from the cerebellar substance; about one-third part of the white matter of the posterior part of the lobe was destroyed and replaced by the growth, but very little of the grey matter was destroyed, as it was flattened out in a thin layer over the surface of the tumour. It occupied the cerebellar fossa. The first symptom was giddiness, which long preceded all others, and was worse on movement. Then came headache in the left occipital region and at the back of both orbits. The patient suffered from attacks of giddiness, ending in vomiting, faintness, and palpitation. His gait was uncertain, inclined to be staggering in a bad light, but he did not especially tend to fall to one or other side; the legs were weak; the knee-jerks were diminished, the right was greater than the left. There was mild optic neuritis, most marked in the right eye. Pupils dilated, left larger than right. Mentally he became more dull as the illness proceeded. The right arm was a little weaker than the left.

Later in the illness optic neuritis and consequent failure of sight increased, the visual fields showed moderate concentric contraction; there was slight deafness; the gait became weaker and more tottering; there was no definite paralysis, but all the limbs were weak, this weakness was most evident in the muscles of the legs and back. Pupils remained the same; the knee-jerks became more active, that on the right side becoming exaggerated.

An operation undertaken to relieve pressure symptoms by making a large trephine opening in the skull and incising the dura mater as a palliative measure in the absence of definite localising symptoms, unfortunately resulted in the formation of a large hernia cerebri, and he died four weeks afterwards.

Case III.—A boy, aged 6 years; a tubercular tumour the size of a large walnut, with a firm, yellow, caseous centre, occupied the central white matter of the *left* cerebellar hemisphere, and extended to its surface on the inferior aspect of this lobe. The other lobes of the cerebellum were normal. The cerebellar peduncles were not especially involved by the growth, but the middle peduncle appeared to be smaller on the left side.

The first symptoms were frontal headache, most marked on the right (opposite) side, and attacks of giddiness with vomiting, during which he was unable to stand; later the headache was also in the occipital region; there was no local tenderness. No mental symptoms were present. The gait was staggering; the direction of staggering was to the right, with a tendency to fall to this side. The muscles of the back and of the trunk were weak, and there was an oscillating tremor of the body and head on first moving. There was weakness and tremor of the right arm, but no absolute paralysis of the limbs. The right knee-jerk was normal, the left diminished. Double optic neuritis was present, most intense on the left side. The left pupil was larger than the right.

Later, seven and a half weeks before death, the power of standing and walking was lost; when the patient tried to stand he fell either backwards or to the right. During one attack of vomiting the head was retracted and the body in position of marked opisthotonos. The tremor in the right arm increased, was jerky, and somewhat more pronounced on movement. The left arm and leg were now somewhat weaker than the right, but paralysis affected the legs chiefly. The right knee-jerk was exaggerated, and there was ankle clonus on this side. Headache was in the occipital and right frontal regions. Both knee-jerks disappeared just before the final stage; symptoms of meningitis were present ten days before death in coma.

The knee-jerks were thus only lost at the end, as was also the case in the first patient; in both cases i. and iii. falling or staggering was to the side opposite the growth; opisthotonus in extreme degree was present as a terminal symptom in one, and as a transient phenomenon in the other. There was occipital headache in all three, frontal in two, distinctly confined to one side, the opposite, in one only, in the second case it was at the back of the orbits.

In the second and third cases the legs were chiefly affected with weakness, and there was distinct weakness of the muscles of the back. In the third case there was evidence of pressure on the pyramidal tract on the same side, leading to exaggerated reflexes and tremor in the opposite limbs, but at one period the loss of power was more marked in the arm of the same than that of the opposite side.

In the first case the symptoms corresponded in some respects to those observed by Dr. Acland and Mr. Ballance[1] in a case of cerebellar abscess. Thus the patient lay curled up in bed on the side opposite the lesion, with the limbs flexed, and the side of the face corresponding to the side of the lesion uppermost; there was rotation to the opposite side in the longitudinal axis of the body (screw in), deviation of the eyes to the opposite side, and great loss of power in the lower extremities.

The second case is further of interest as indicating the symptoms due to a very slowly-produced, non-irritating lesion (growth) affecting the lower part of one lateral lobe, and showing that such a tumour may exist in this situation without giving any sufficient indications as to the side affected. It also shows the importance of giddiness with slight optic neuritis as a very early symptom in such a case. It would perhaps go to support the view expressed by Dr. Acland and Mr. Ballance[1] that to produce symptoms due to interference with the fronto-cerebellar tract of fibres the lesion must either occupy the anterior and inner part of the lateral lobe or destroy nearly the whole lateral lobe.

The following case is a good example of distension of one lateral ventricle at its posterior part. The patient was a man, aged 25. On removing the brain *post-mortem*, a lobulated tumour about 3 inches long, 1½ inches in breadth, and 1½ inches in thickness was found growing from the roof and internal aspect of the posterior horn at its junction with the body of the right lateral ventricle. The descending and posterior horns were much distended with fluid from the tumour obstructing the connection with the rest of the ventricles, the white matter being spread out and thinned, but not otherwise abnormal. The hippocampal and uncinate gyri at the free inner margin of the temporo-sphenoidal lobe were represented by a bulging, thin-walled, translucent membrane, and it seemed probable, though it could not be certainly made out after removal of the brain, that the much distended tip of the descending horn of the lateral ventricle had pressed upon the right 5th nerve.

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[1] *St. Thomas's Hospital Rep.*, 1894.

The symptoms began with intense paroxysmal headache over the vertex of the skull, followed by loss of feeling over the right side of face. The patient was morose and ill-tempered, mentally dull and apathetic, and slow in speaking. Pulse 60 in rate, dropping sometimes to 48. No facial paralysis. Paralysis of the right 5th nerve, at first of the sensory, later of the motor portion also; the anæsthesia ended about ½ inch, about the line of lower jaw. Smell and taste slightly deficient on right side; hearing normal.

Pupils normal; acted well, but sluggishly; no nystagmus; visual fields normal; no optic neuritis. Speech and swallowing normal; gait normal; no muscular paralysis, rigidity, or wasting. No ataxy of movement. Superficial reflexes and knee-jerks normal. No anæsthesia elsewhere than in the distribution of right 5th nerve. A month before his death he suffered during a period of eleven days from paroxysmal attacks of trismus, coming on every few minutes; these attacks then ceased. For two or three days before his death there were attacks of spasmodic contraction of the muscles of the back of the head and neck; no opisthotonos; the optic discs showed now commencing swelling, with blurring of their edges. There were no other symptoms.

The distension of the posterior part of the ventricle was extreme, owing to the position of the tumour completely cutting off its connection with the rest of the ventricle; a prominent symptom was the paralysis of the sensory branch of the 5th and irritation followed by paralysis of the motor portion. Possibly this affection of the 5th might be useful in diagnosis in future cases of the same kind.

In a case of tumour of the lateral ventricle, a large, much lobulated growth, 2½ inches in length by 3 inches in breadth, formed the floor of the left lateral ventricle; the right optic thalamus, corpus striatum and anterior three-fourths of the corpus callosum were undisturbed; behind the latter the lobes of the growth appeared, and the posterior margin of the corpus callosum was softened. The corpora quadrigemina were rather flattened from pressure, and the left crus cerebri pushed forwards. At the free inner edge of the temporo-sphenoidal lobe from the distension of the ventricle the hippocampus major was protruded and everted.

The chief symptoms were vertigo; severe attacks of vomiting; deep-seated, gnawing pain in the back of the head and over the vertex; some deafness and tinnitus aurium. Expression vacant; memory bad; power of concentration or sustained thought much impaired; dull and apathetic. Speech slow, and articulation indistinct. Staggering gait, staggering to either side, most to right; difficulty in balancing the head; no actual paralysis; no loss of sensation; double optic neuritis, leading to loss of sight. There was partial hemiplegia on the right side lasting for some months about a year before death, but this passed off, and did not recur.

The last case I have to mention is one that I owe to the kindness of my colleague, Dr. Markham Skerritt. Unfortunately there are no notes of the case for a period of two months before death, but they are full up to that time, and the symptoms somewhat remarkable. The duration of the illness was five months, the patient was a girl, aged 16, and the first symptoms were severe frontal headache, with a feeling as if "her head were stuffed up from a severe cold;" pain in the back, causing some difficulty in walking; there was no staggering gait and no paralysis in the limbs; fits, general in character, in which both upper and lower extremities were flexed at all joints, except the fingers, which were extended; consciousness was not completely lost, the surface of the body was at first blue and cold, and the fits were followed by profuse sweating, with violent crying and sobbing.

Three months from the onset and two before the fatal termination her legs were weak, so that she stayed in bed, but there was no absolute paralysis, and muscular power in all other parts of the body was good; there was no ataxy of movement; no rigidity of muscles. She lay on her right side in bed, with the lower extremities curled up. Sensation was unaffected. There was no oculo-motor paralysis; the pupils reacted feebly to light, normally to accommodation; the right pupil was dilated. The right knee-jerk was absent, the left normal; superficial reflexes normal. There was intense optic neuritis, most marked in the right eye, which soon became blind. Hearing, taste, and smell normal. She was dull, listless, and apathetic, and some failure of mental power was noticed (loss of memory). She suffered from fits, which had now changed in character, consciousness being completely lost; the limbs were drawn up and rigidly flexed, but there were no clonic spasms; in the fits the bladder was emptied and the pulse became slow and almost imperceptible. They were followed by crying and sobbing and by great prostration.

Post-mortem.—A soft glioma, the size of a large walnut, into which hæmorrhage had occurred, occupied the superior medullary velum, had destroyed and replaced the corpora quadrigemina and pineal gland, and extended a short distance upwards into the great transverse fissure of the brain, lying beneath the choroid plexuses, and downwards a little way into the middle lobe of the cerebellum. The lateral ventricles were moderately distended.

Although the notes were for some reason not continued to the end of the illness, it seems worthy of note that three months from the onset and two months before death, when symptoms of an intra-cranial tumour were sufficiently well marked, and vision in the right eye was lost from the intensity of the optic neuritis, a tumour in this region should neither give rise to oculo-motor paralysis of any kind, nor to ataxy of gait or movement. By most writers ophthalmoplegia is regarded as an early symptom of tumours of the corpora quadrigemina, though Nothnagel stated[1] that defect of co-ordination in walking is the earlier sign. The loss of the right knee-jerk is interesting; loss of the knee-jerks has been noticed in other cases.[2]

In conclusion, it seems that allowance must be made for variation in the symptoms produced by intra-cranial growths in identical parts of the brain in different cases. One reason for this variation lies in the difference in structure or consistence of the various kinds of pathological new formations, and also in their rate of growth; whether the tumour is slow or quick-growing, and whether it tends to infiltrate itself into the surrounding tissues, or has no such tendency, and destroys them from pressure. In the latter case a slow-growing tumour may attain a considerable size but produce few symptoms, because the surrounding parts have time to adjust themselves to their new conditions, so that next to accurate localisation, or rather as an aid to it, the diagnosis of the nature of the growth is exceedingly important, but unfortunately often most difficult or impracticable.

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[1] BRAIN, 1889.

[2] Drs. W. A. Turner and Guthrie, *Lancet*, 1895, vol. i., p. 274, and Dr. W. B. Ransom, *Ibid.*, p. 1115.

III.—SEYMOUR J. SHARKEY, M.D., F.R.C.P.

ON considering how I could best respond to the invitation given me by the Council of the Neurological Society, to take part in the discussion to be introduced by Dr. Beevor, I came to the conclusion that I would analyse the phenomena presented by patients suffering from tumour in the regions indicated, who had been admitted into St. Thomas's Hospital and had died there while I had been physician.

I have to return my best thanks to my colleagues who kindly allowed me to make use of their cases, and to Dr. Russell, the medical registrar, who was good enough to collect the notes for me.

The period of time covered is from 1890 to 1898, and the list of cases is as follows :—

Under my care	11	21
Under Dr. Bristowe, Dr. Ord, Dr. Acland and Dr. Payne......	10	

Frontal Lobe	8
Occipital Lobe	1
Temporo-sphenoidal Lobe	2
Temporo-sphenoidal and Occipital	1
Corpus Callosum	1
Lateral Ventricle	1
Optic Thalamus	2
Optic Thalamus and part of Caudate Nucleus ...	1
Cerebellum	4
	21

1.—TUMOUR OF RIGHT FRONTAL LOBE. (DR. PAYNE.)

L. H., aged 27, *admitted December* 17, 1897, died May 23, 1898. Illness commenced in August, 1897. Headache, especially in forehead and right orbit ; vomiting ; *squint of right eye* (turned outwards) ; double vision.

On admission.—Left internal strabismus ; double optic neuritis ; no paralysis ; *conjugate movements of eyes deficient ;* moderate proptosis.

Later.—Definite weakness of left leg and arm; lethargic; movements of eyes of frequent occurrence; no lateral or vertical nystagmus.

Later.—Anosmia—first noticed by herself; increasing left hemiplegia; no loss of sensation at any time; no convulsions. *Towards end she had visual hallucinations, and wandered.*

Post-mortem.

Membranes adherent to right frontal lobe; brain caseous on section; right frontal lobe occupied by a large growth; both olfactory nerves flattened; growth extended backwards as far as ascending frontal; extreme anterior end of internal capsule involved.

2.—TUMOUR OF FALX CEREBRI INVOLVING LEFT FRONTAL LOBE. (DR. BRISTOWE.)

E. H., aged 63, *admitted August* 31, died *December* 16, 1891. Fifteen months before admission left breast removed for carcinoma. *Loss of memory for words and loss of speech; mental failure; loss of power of writing; weakness of right hand*, then of right foot, and mouth; rigidity of right leg; no optic neuritis (up to October; no later mention of it).

Post-mortem.

Tumour of size of tangerine orange sprang from left side of falx, where it was attached to crista galli. It had displaced and removed the whole of anterior part of frontal lobe. It shelled out easily.

3.—TUMOUR OF LEFT FRONTAL LOBE. (DR. ORD.)

M. M., aged 45, admitted April 22, died June 9, 1895. Onset six weeks before admission, with attack of vomiting and unconsciousness. Next fourteen days *inability to speak or understand what was said; attacks of cramp and convulsive movements of right arm and leg;* headache; optic neuritis, mainly in left eye; *paralysis of right side of face; weakness in right arm and leg; no alteration in sensation; quiet and drowsy, and very dull mentally; saw and talked to imaginary persons.*

Post-mortem.

Tumour involving left lower frontal convolution and greater part of left frontal lobe (glioma).

4.—Cyst of Right Frontal Lobe. (Dr. Payne.)

G. S., aged 31, admitted October 1, died December 23, 1896. Fits, in which *left arm and left side of mouth moved;* headache; vomiting; optic neuritis; *some drooping of left eyelid; diplopia; slight loss of power down left side; drowsy and apathetic* (after trephining).

Post-mortem.

Cyst of right frontal lobe, below level of corpus callosum; dura mater adherent over it.

5.—Tumour of Right Frontal Region. (Dr. Acland.)

A. W., aged 22, admitted October 5, died November 1, 1894. Fits up to age 14. Eight days before admission a fit, since which *she had scarcely spoken. Complete paralysis of right side—arm, leg, and face; paralysis of external rectus on right;* some impairment of sensation in right hand and arm; *mentally very dull;* headache; optic neuritis.

Later.—Left external rectus affected; coma.

Post-mortem.

Large tumour in right frontal region, occupying its whole extent. Left motor tract appeared quite healthy, as did pons, medulla, and cerebellum. Decussation of pyramids normal.

6.—Tumours of Right and Left Frontal Lobes.

M. D., aged 40, admitted May 23, died September 12, 1892. Five years before one fit. Headache one year before admission; failure of sight. *Hallucinations of sight followed* by one fit six months before admission.

After admission.—Frequent fits and movements of right arm and leg; stertorous breathing, with unconsciousness; some loss of sensation in right conjunctiva and right side of face; double optic neuritis; no hemianopsia; *delirious at times, with hallucinations.*

Later.—Left arm and leg obviously weaker than right; definite anæsthesia of left side of face; blind; right knee-jerk absent, left very feeble; gradual coma.

Post-mortem.

In *left hemisphere* growth occupied upper, outer, anterior, and under aspects of frontal lobe, reaching nearly to anterior limit of

Sylvian fissure = 1½ in. × 1½ in. Brain substance around much softened.

In *right hemisphere* similar tumour, 1½ in. × 1½ in., occupying white matter of frontal lobe, beginning 1 in. from tip and stopping posteriorly just short of caudate and lenticular nuclei.

7.—Glioma of Left Mid-Frontal Region.

M. M., aged 25, admitted March 21, 1893, died May, 2, 1894. In October, 1886, in-patient, with fits, vomiting, optic neuritis.

On admission in 1893.—Both arms weak, equally ; left leg weaker than right, and smaller ; no anæsthesia or hyperæsthesia ; smell and taste normal ; knee-jerks increased ; double optic neuritis ; *weakness of both external recti ;* general tenderness over head and severe headache ; *speech slow and scanned ; many* fits, which generally began with *twitching of mouth, and in all she lost speech ;* and this lasted for a short time after fit. In fits right arm and right side of mouth more frequently affected than left, and *conjugate deviation of head and eyes* as often to one side as the other. Became gradually more drowsy, and died suddenly.

Post-mortem.

Tumour of mid-frontal region on left, causing bulging on internal aspect of hemisphere. Another small tumour at posterior part of convolution of corpus callosum on left.

8.—Glioma of Left Frontal Lobe.

L. A. R., aged 17, admitted July 27, died August 3, 1895. Quite well till day of admission, then found semi-conscious in her room.

On admission.—Almost unconscious ; arms and legs somewhat rigid ; slight optic neuritis ; remained unconscious till death.

Post-mortem.

Hæmorrhage had occurred from tumour into whole left subdural space.

9.—Tubercular Mass in Right Temporo-Sphenoidal Lobe.

M. D., aged 38, admitted December 2, died December 5, 1890. Pain in head ; vomiting ; drowsy and wandering at times ; irritable temper ; optic neuritis ; coma.

Post-mortem.

Mass of size of walnut in outer and hinder part of right temporo-sphenoidal lobe.

10.—TUMOUR OF LEFT TEMPORO-SPHENOIDAL LOBE. (DR. ACLAND.)

M. C., aged 37, admitted November 6, died November 27, 1895. Five years before first fit, many after that. For ten weeks before admission pain in head and limbs; occasional vomiting; some loss of memory; optic neuritis; no paralysis or anæsthesia; right pupil rather larger than left; nystagmus during last few days.

Post-mortem.

Tumour in left temporo-sphenoidal lobe, extending from before backwards nearly the whole length of the lobe.

11.—GLIOMA OF LEFT OCCIPITAL LOBE.

A. G. K., aged 18, admitted July 29, died August 2, 1897. Severe headache; vomiting; pupils dilated; double optic neuritis; grasp of right hand somewhat less than left; sensation normal; knee-jerks obtained with difficulty; retraction of head; refusal of food.

Post-mortem.

Encapsuled growth, 2 in. × 1 in., at tip of left occipital lobe above posterior cornu of lateral ventricle.

12.—TUMOUR AT JUNCTION OF LEFT TEMPORO-SPHENOIDAL AND OCCIPITAL LOBE. (DR. PAYNE.)

S. P., admitted December 18, 1889, died February 3, 1890.

In February.—Three "fainting fits;" pain in head; vomiting; optic neuritis; wasting and weakness of left arm, followed by weakness of left leg, also paralysis of left side of face; no loss of sensation; no rigidity; knee-jerks brisk, and ankle clonus; tremor, and some inco-ordination of right arm; gradual coma.

Post-mortem.

Recent basal meningitis. Tumour as large as a walnut at junction of 2nd and 3rd temporal convolutions with occipital lobe (superior temporal convolution and angular gyrus *not* involved). Tumour did not nearly reach internal capsule, but changes and yellow tinging were noticed in external capsule, posterior part of internal capsule, and part of optic thalamus.

No disease of *right* hemisphere, pons, medulla, crura cerebri, or cerebellum.

13.—Tumour of Corpus Callosum.

E. C., aged 46, admitted December 15, 1897, died April 25, 1898. In 1893, cancer of breast; operated on.

September, 1897.—Vomiting; headache; convulsions and unconsciousness; *got childish and comatose;* no sensory affection; *gradual paralysis and more or less rigidity of limbs; five months comatose, like person in deep sleep;* no optic neuritis.

Post-mortem.

Tumour commencing at anterior extremity of corpus callosum and growing backwards above ventricles.

14.—Glioma of Right Lateral Ventricle.

F. W., aged 21, admitted August 16, 1895, died January 19, 1896. Headache, chiefly frontal; vomiting; unconsciousness three days; optic neuritis; giddiness; no paralysis, motor or sensory; drowsiness; wandering; pain in left ear; tenderness on percussion over left mastoid and occipital region; pain and stiffness in back of neck.

Tumour grew from anterior extremity of the right lateral ventricle, the latter being much distended with fluid.

15.—Tumour of Right Optic Thalamus.

F. M., aged 22, admitted January 25, died January 30, 1897. Headache; sickness; vomiting; pain and numbness on left side of body.

On admission.—Unintelligent; right arm weaker than left; sensation normal; increased knee-jerks and tendency to clonus; fits, generalised or localised; optic neuritis; coma deepening; left external strabismus; Cheyne-Stokes respiration.

Post-mortem.

Tumour occupying outer two-thirds right optic thalamus, also smaller tumour in same position on left.

16.—TUMOUR OF LEFT OPTIC THALAMUS. (DR. BRISTOWE.)

H. J., aged 18 months, admitted April 17, died June 29, 1890. Three months before admission head increasing in size; could then stand when holding chair, but gradually lost all power in legs; optic neuritis; weakness of external recti and increasing drowsiness; rigidity of limbs; finally retraction of head.

Post-mortem.

Ventricular distension great; no meningitis; large tumour involving posterior part of left optic thalamus and anterior corpora quadrigemina. It had the size and shape of two walnuts in contact.

17.—TUMOUR OF RIGHT OPTIC THALAMUS AND CAUDATE NUCLEUS. (DR. PAYNE.)

T. W., aged 24, admitted August 29, 1892, died January 13, 1893. Illness began two months before admission. Weakness and pain in left leg; weakness of left arm; no anæsthesia; pain in right side of forehead and neck; optic neuritis; vomiting; coma.

Post-mortem.

Into right lateral ventricle projected a large, yellowish mass, which arose from and concealed the entire right optic thalamus and the posterior quarter of the caudate nucleus. The mass was as large as a tangerine orange. Right crus cerebri was markedly flattened, and the greater part of the lenticulo-optic part of internal capsule was involved.

18.—TUBERCULAR MASS IN CEREBELLUM.

A. W., aged 7, admitted February, died July, 1897. Began three-and-a-half years ago. Fits; no headache; vomiting.

On admission.—Great unsteadiness of gait; no loss of muscular power; (?) right arm more awkward than left; *slight lateral nystagmus;* double optic neuritis; *three fits, with rigidity of right arm in one,* of legs in others; inco-ordination more marked—required to be led; drowsy; *weakness of conjugate deviation of eyes to right;* Cheyne-Stokes respiration.

Post-mortem.

Great distension of ventricle; right lobe of cerebellum converted into a firm caseous mass; middle lobe thinned and torn by ventricular distension and partly possibly involved in the mass.

19.—Cerebellar Tumour.

W. S., aged 28, admitted September 3, died September 7, 1890. Headache; giddiness; vomiting; delusions; irritable; violent; optic neuritis; speech slow and mumbling; tongue protruded to right; writing shaky.

On day of death.—Retraction of neck.

Post-mortem.

In vermiform process, and extending into each lateral lobe, was a cavity of the size of a walnut, containing fluid and soft substance.

20.—Glioma of Cerebellum.

W. H., aged 9, duration two years. Vomiting; giddiness; *staggering* (tendency to left); headache (occipital); *lateral nystagmus;* (late) optic neuritis; *weakness of external recti.*

Towards end.—Left hemiplegic weakness; drowsiness.

Post-mortem.

Ventricles much dilated. Infiltrating tumour and hæmorrhage involving middle and both lateral lobes of cerebellum.

21.—Cystic Tumour in Cerebellum. (Dr. Acland.)

M. J. B., aged 38, admitted November 12, died December 3, 1896. Six weeks before admission illness began. Pain in back of head; double optic neuritis; giddiness; vomiting; *drooping of left upper eyelid;* apathetic; *left eye moved with difficulty into outer canthus;* no anæsthesia; drowsy.

Post-mortem.

In posterior half of right hemisphere of cerebellum was cyst, with yellowish, mucoid contents; dentate nucleus had disappeared; into cyst at one spot projected a reddish, vascular growth; moderate dilatation of ventricles.

General Symptoms of Intra-Cranial Tumours.

The usual symptoms which indicate the presence of intra-cranial tumours are one or more of the following:—Pain in the head; vomiting; giddiness; optic neuritis; convulsions; paralysis—motor or sensory—of cerebral type, or of cranial nerves.

On these we rely for our diagnosis that a tumour exists. Can we go further and say that it is in one or other of the regions suggested by Dr. Beevor? Before looking more critically at the symptoms which were present in the cases I have analysed, one ought to answer the question whether one can with certainty diagnose the hemisphere in which the tumour is. The position of pain is unreliable as a guide to the position of the tumour, so much so that one of the cases of frontal lobe tumour was trephined on the wrong side.

Unilateral optic neuritis is rare, but when it occurs, it is generally on the diseased side.

We might at any rate expect that if hemiplegia was present, we could hardly go wrong. But cases 5 and 12 show that not even this is a certain guide, for the hemiplegia which occurred in them was on the same side as the disease.

As regards paralysis of cranial nerves, only the olfactory and 7th and 8th are of much value, paralysis of the former pointing to disease in the frontal lobe or tip of the temporo-sphenoidal, paralysis of the two latter usually pointing to tumour in the corresponding half of the cerebellum or its neighbourhood.

Tumours of the frontal lobe may exist, even when very large, without producing any serious symptoms for a long time. The most usual symptoms in my cases were

(1) Convulsions or paralysis of hemiplegic type, with or without some loss of sensation.

(2) Aphasia, if left side was the seat of disease.

(3) Mental symptoms, especially lethargy, mental dulness, hallucinations, perhaps most frequently of sight.

(4) Various affections of the movements of the eyes.

(5) Anosmia.

Temporo-sphenoidal lobe.—The cases show that a tumour in this lobe, even when large, may have no localising symptoms.

Tumours of occipital lobe may have no localising symptoms.

Hemianopsia, if present, is an important sypmtom, although it would not prove that the tumour was in the occipital lobe.

It is a question whether anorexia and refusal of food are not suggestive of disease of this lobe.

Tumours of the corpus callosum.—The late Dr. Bristowe pointed out in an article in Brain, vol. vii., that the symptoms characteristic of tumour of the corpus callosum are gradual onset of hemiplegia, often bilateral, stupidity, drowsiness, cessation of speech, difficulty of getting food down the throat, absence of implication of the oculomotor or other cerebral nerves.

Coma.—In the case I have just brought to your notice, the patient remained comatose for five months, the coma suppressing the symptoms of paralysis which had developed, except the rigidity of limbs. The picture of placid coma which she presented, extending over months, was such as I have never seen, except in tumours of the corpus callosum.

Lateral ventricle.—The case recorded presented no localising symptoms.

Tumours of the optic thalamus.—The strabismus which may occur is probably due to pressure of the tumour on the nerves at the base of the brain, and the paralysis of limbs to the involvement of the internal capsule. Apparently, tumours in the optic thalamus present no symptoms which guide us surely to their position, but if the tumour is a slowly growing, infiltrating mass, there may be phenomena which strongly suggest the locality of the disease, viz., the gradual supervention of tremors, paralysis, and finally rigidity of limbs, first on one side, then on the other, due to a gradual invasion of the internal capsules (v. Gulstonian Lectures, 1886).

Cerebellum.—Tumours may exist in the cerebellum, especially in the lateral lobes, without giving any evidence of their presence. But the phenomena which come most prominently forward in my cases are unsteadiness of gait, without loss of muscular power; some affection of the movements of the eyes—nystagmus—weakness of conjugate movements—weakness of individual muscles; attacks of rigidity of limbs.

IV.—G. Newton Pitt, M.D., F.R.C.P.

The reports of fifty-eight cases of cerebral tumours which have been examined in the *post-mortem* room at Guy's Hospital during the past fourteen years have been consulted, and I have to thank my colleagues for their kind permission to make use of their cases. In many instances the extent of the growth was so extensive and its limits so ill-defined that no useful deductions could be drawn from the symptoms which had been noted.

Cerebellar Tumours.

Out of fourteen cases of cerebellar tumours three were multiple, and one involved the middle lobe projecting into the iter. With tumours involving the lateral lobes vomiting was a more or less marked symptom in all.

Giddiness occurred in six, and in six also the gait was staggering; in four the stupor was extreme. Once there was marked weakness in the muscles of the back, but this probably was also present in some of the other cases where the patient could hardly stand. Once, there was marked general loss of power, and once, the limbs on the same side of the tumour were very feeble. In this case, the corresponding knee-jerk was absent. In four cases, the knee-jerks were noted to be absent; and in four, exaggerated. Optic neuritis was only absent in two cases, and in one of these it commenced to develop just before death. The position of the headache varied, being usually either occipital or frontal, but in one case it was also temporal, and in another it extended down the neck. The pain in the occiput was chiefly on the same side as the tumour, while when the pain was in the frontal region, to which it was twice limited, it was on the opposite side. The explanation which is offered being that the fibres from the frontal region to the corpus dentatum of the cerebellum of the opposite side, form the path of communication.

There is still uncertainty as to which side the patient generally falls; in three the patient fell to the same side as the tumour, in one he tended to fall to the same side and backwards; while once the patient tended constantly to turn round to the same side as the tumour. In none was the movement to the opposite side.

When there was pressure on the iter, stupor developed, the speech and actions were very sluggish, one patient yawned frequently. The remarkable way in which some of the patients put on fat was very striking. The optic neuritis and the headache appear to be more intense and to develop earlier than is the case with tumours situated elsewhere. Nystagmus was not infrequent and diplopia was noted twice; once the fifth and once the seventh nerves were affected.

FRONTAL.

Ten tumours involved the frontal region, the greater part of the growth being in most of them, intra-medullary. The features of special note were in four cases, the marked mental torpor; in one case, this lasted for six months, the patient's condition being one of absolute mental apathy, and in some cases this passed on to coma. Incontinence of urine also occurred in four cases, and was probably due to the mental condition. The headache in one case was occipital, and in most was not limited to the frontal region. The vomiting was neither so constant nor so frequent as occurs with tumours elsewhere, for example in the cerebellum, nor was the headache so severe.

The optic neuritis tended to involve the affected side more than the opposite. In two cases there was rigidity, and in one case stiffness of the neck.

In one patient the head tended to turn to the side of the lesion, and in another at one period of the illness there was conjugate deviation of the eyes to the affected side.

The knee-jerks were exaggerated in some cases and absent in others.

Two patients had fits, but in more than one case there were no localising symptoms.

Of the local paralyses there was in one, ptosis of the eyelid on the same side and in another there was weakness of the opposite side of the face.

None of the tumours were limited to the frontal cortex.

Corpus Callosum.

In two cases the corpus callosum was involved; in one the anterior third was affected, but the symptoms were not characteristic, the patient lying in a state of stupor with rigidity for months.

In the other case the patient was in a state of stupor, and had a fit, commencing in one hand. There was frontal headache, vomiting, double optic neuritis, with increasing coma. The illness was of short duration, and there was not sufficient evidence to enable the diagnosis as to its site to be made. The tumour was a large one, involving the corpus callosum, and extending into both hemispheres.

Uncinate.

Two tumours which involved the *uncinate gyrus*, gave rise to hallucinations of smell of a most marked character. In one case, in which there was also optic neuritis, headache, vomiting, and epileptiform convulsions, an accurate diagnosis of the locality of the tumour was made during life. In the other case the growth was of an extensive character, and gave rise also to hemiplegia, &c.

Angular and Supra-Marginal.

Of four cases, involving the angular and supra-marginal regions, in one case the hemiopia and optic atrophy enabled a correct diagnosis to be made; while in the others the mental condition of the patient interfered with any observation as to the field of vision being made.

Occipital.

Two tumours were limited to the occipital region invading the cortex, but no symptoms enabling the mischief to be localised were noted, nor were the fields of vision mapped out, owing to the fact that in one case the patient was admitted for a carcinoma of the breast, which was removed, and the tumour was not suspected. In the second, the patient was admitted in a state of stupor, so that she was incapable of answering satisfactorily. Four other large tumours involving the centrum ovale, extended into the occipital region.

In three cases the tumours were not diagnosed, one being a case of cancer of the breast, the first symptom being a fit which proved fatal, the second being a case of uncontrollable vomiting, and the third of meningitis.

In one or more, the evidence pointed to a tumour on the opposite side to that on which it was found.

Optic neuritis develops most rapidly with cerebellar tumours, and more slowly when the tumour is in the centrum ovale.

The rapidity with which a growth develops largely influences the symptoms, and determines the severity of the optic neuritis and of the headache, and also the frequency with which symptoms due to pressure on distant parts, develop.

V.—Marcus Gunn, M.B., F.R.C.S.

When invited to take part in this discussion, I was told that I should be expected to pay attention to the localising value of optic neuritis in cases of intra-cranial tumour, and to the question of the side on which the neuritis first occurs, or is most marked, relative to the position of the tumour. I shall accordingly confine my remarks to these parts of the enquiry.

I do not propose giving fresh statistics as to the frequency of optic neuritis in the case of intra-cranial tumours in different positions, but I have examined several of the most important statistical tables that have been published bearing on the subject, and have analysed them with due reference to the present discussion. My thanks are due to Dr. James Taylor for furnishing me with important observations on a series of fifty-eight cases, which he had analysed as to the presence or absence of optic neuritis, and as to the degree of neuritis in each optic nerve in reference to the side of the brain on which the tumour was supposed or proved to be. I have also to thank Dr. Beevor for kindly giving me references to any points likely to prove useful to me in the cases that he has brought forward to-night.

The outstanding facts of these investigations, and of all clinical experience are, first, the great frequency of optic neuritis as a symptom in connection with tumour in any of the positions mentioned by Dr. Beevor; secondly, the particular proneness of the optic nerve to be affected in tumour of the cerebellum, and, after it, in tumour of the basal ganglia, and of the occipital, frontal, and temporo-sphenoidal lobes of the cerebrum—these being mentioned here in the order of greater frequency.

Such facts are, however, evidently of practically no localising importance in any particular case. Indeed, common as optic neuritis is in intra-cranial tumour, we know that a large new growth may exist, and be found after death, without any optic neuritis having been present; while, on the other hand, optic neuritis has been diagnosed during life, without any cause for its occurrence being recognised by highly competent observers at the autopsy.

It is not so much the locality of an intra-cranial tumour, as its behaviour, that influences such symptoms as headache and optic neuritis. Rapid growth or liability to sudden congestion; association with an increase of cerebro-spinal fluid; or a complication with basal meningitis, are the facts that seem to particularly predispose to the occurrence, and afterwards to the severity, of inflammation of the optic nerve. It is doubtless in consequence of this relation between behaviour and intensity, and not from anything particular in the mere position of the new growth, that tumours affecting the cerebellum peculiarly predispose to involve the optic nerve.

Such being the case, is there anything in the character and behaviour of the optic neuritis itself—apart from its mere occurrence—that is of any localising value? I know that I have the support of some of my medical colleagues at Queen Square in believing that cerebellar tumour very often excites a peculiarly intense optic neuritis, with great engorgement of the papillæ, and marked œdema of the surrounding retina, with, not uncommonly, a macular stellate figure similar to what we get in so-called albuminuric retinitis. This latter observation has, I know, been made independently by Dr. Jas. Taylor. I have elsewhere offered an explanation of this retinal condition, reconciling the liability to its occurrence from very different causes, and all I need say here is that, as regards all forms of intracranial disorder, this retinal appearance with intense papillitis is in my experience most apt to occur in cerebellar tumour and in basal meningitis—perhaps in cerebellar tumour because it may so readily cause a basal meningitis. I have met with this form, however, in at least two cases of frontal tumour, in neither of which, I believe, was there any evidence of the basal meninges being involved.

We know that, in some cases of optic neuritis, there is a markedly rapid increase in the amount of swelling of the papillæ, presumably due to a concurrent increase of intracranial pressure—for the converse at least is true, that a surgical relief of intra-cranial pressure is followed by, and doubtless causes, a similarly rapid diminution of disc-prominence. If it can be shown that tumours in certain localities are particularly liable to raise intra-cranial tension, then this ophthalmoscopic sign would have some localising value, but not otherwise.

And now a few words as to a possible relationship between uniocular optic neuritis and one-sided brain tumour. The explanation of optic neuritis accompanying intra-cranial tumour is confessedly still obscure, and it is difficult to realise why, on any of the theories advanced, one optic nerve should be attacked and the other long remain normal. But unilateral *excess* of neuritis may admit of explanation.

First.—If optic neuritis be of the nature of a gradually extending inflammation, communicated along the pial sheath of the nerve from the basal meninges, a greater proximity of the exciting tumour to one optic foramen would cause the optic nerve passing through it to be first involved. If this reasoning be correct, in the case of a one-sided growth affecting the base far forwards in the frontal and anterior part of the temporo-sphenoidal lobes, the optic neuritis would be first recognised, and would for some time be in excess, on the same side as the tumour. In the case of a tumour located further back, the affection of the pia mater would presumably extend forwards equally along both nerves after it had reached the chiasma; and in such a case, accordingly, the optic neuritis might be expected to appear practically simultaneously, and be equal in intensity, on the two sides, and that, in any case, it would have no lateral localising significance. So much for theory. Statistics lend considerable support to this reasoning. Taking the important analysis of 601 cases of tumour, made by Dr. J. M. Martin last year,[1] and paying attention to the relation between position, occurrence of optic neuritis, and difference of neuritis on the two sides, I find that there was unilateral neuritis (or excess) in ten cases of frontal tumour, and in eight of these it occurred on the *same* side as the tumour. In four cases of neuritis difference, in cases of temporo-sphenoidal growths, the one-sided excess was always on the *same* side. Putting these together, we have the unilateral excess of neuritis on the same side as the tumour in twelve cases out of fourteen.

How now as to the cases of tumour in the parieto-occipital lobes and in the cerebellum? The unilateral character of the neuritis was noted in ten such cases altogether; in *five* of these it was on the *same* side as the tumour, and in *five* on the opposite side. Statistics are dangerous, I know, but these facts are certainly striking. (It will be found that these are not quite the conclusions drawn by the author quoted, as he considers *all the cases of tumour together* in reference to this question of side.)

Another interesting statistical result bearing upon this argument is, that a one-sided difference of neuritis proves to be much more common in tumours of the cerebral lobes than in tumours of the cerebellum; it occurred in 20 per cent. of all the instances of neuritis with tumour of cerebrum; in only about 3 per cent. of those associated with tumour of cerebellum. This is, of course, in favour of its being communicated by contiguity.

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[1] *The Lancet*, July 10, 1897.

Second.—On the theory of a *descending cerebritis*, it is impossible to understand how the inflammation can fail to be transmitted along both optic nerves on passing the chiasma, even though it had previously been confined to one optic tract. The same remark applies to the supposed influence of the external geniculate body.

Third.—If increased intra-cranial pressure be taken as the determining cause, it is manifest that the actual tension must be the same in all parts of the cranial cavity, and that if the two optic nerves are symmetrically situated they must be equally exposed to the effects of the pressure. But it is possible that anatomical differences, developmental or pathological, may be responsible under pressure-causation for some of the very rare cases where one optic nerve only is inflamed, and of the much more common instances of one nerve being involved in advance or in excess of the other. Minor degrees of want of symmetry of the cranial bones on the two sides are not uncommon. And should the disposition and size of the optic canals be in this way different, it is conceivable that this may determine the escape of one nerve, or a modification of its proclivity to the effects of pressure. But of this form of asymmetry we know nothing exact. In an autopsy on a case of one-eyed neuritis in intra-cranial tumour, it would be well to investigate the possibility of some such anatomical explanation.

Of want of symmetry further forwards, however, we know much more. It is, I think, a fairly general belief, as was pointed out by Gowers, that optic neuritis is, on the whole, more common in hypermetropic eyes. If, with due regard to the frequency of hypermetropia, this is really true, as it seems to be, then it would be interesting to find how far this may be responsible for unequal optic neuritis. In measuring the swelling of the papilla we always require to estimate the refraction of the eye away from the optic disc, at or near the macula by preference, and it is customary from these data to compare the height of the disc above the general fundus-level in the two eyes. But it would be important also always to compare the refraction of the two eyes. How far such a factor is influential, I am not able at present to say; it is unlikely that it should be important except where the difference in refraction is very decided. Of course its influence, if actual, would minimise the localising value of unsymmetrical optic neuritis, restricting its applicability to cases where the refraction of the two eyes is the same or nearly so.

On the whole, then, while we all fully recognise the value of the ophthalmoscope in the diagnosis of intra-cranial tumour, it is necessary to be on our guard not to expect too much from such information, either as to the size of the tumour or its localisation, or disappointment will be the result. Carefully recorded observations, however, may yet teach us much that we wish to know. At present I do not think that we are justified, in respect of this method of localising intra-cranial growths, of saying more than

(1) That intense double optic neuritis, with much swelling and surrounding retinal change, coming on quickly, suggest the *cerebellum*;

(2) That one-sided optic neuritis, or marked difference, suggests the *cerebrum*, and is, on the whole, in favour of the tumour being on the same side as the excess of neuritis, where there are other reasons for localising one in the front of the cerebrum.

VI.—F. W. Mott, M.D., F.R.C.P.

Dr. F. W. Mott referred to three cases of tumour of the 3rd ventricle, which started probably from the pituitary body, and which during life presented very similar symptoms, viz., *recurrent attacks of coma, accompanied sometimes by general or local epileptiform convulsions*, a condition of drowsiness so marked that these patients *would fall asleep* while eating their meals, and in two of the cases this phenomenon was so marked a feature as to be the most prominent and early objective symptom. In one case under his own observation optic neuritis was observed, in another case it is stated distinctly in the notes that the discs were normal, and in the third, where tumour was unsuspected during life, no statement of the condition of the fundus was recorded. All the patients suffered with *headache, but not of a very severe character;* the pain was dull and frontal in situation in two cases. There was *general paresis of the limbs,* but no paralysis, with the exception of slight ptosis of the left eye in one case. The pupils reacted sluggishly to light. The expression was dull, and indicated the mental condition, which was that of *progressive dementia.* In the case under his own care he noted that, although the speech was slow and deliberate, there was no syllabic elision or tremor, nor did the patient show by his writing the characteristic phenomena of general paralysis. This patient, moreover, had an *oscillating gait* like a drunken man. The two cases which occurred in the asylum were looked upon as general paralytics, and the case which was under his care when sent into Charing Cross Hospital was considered by the house-physician to be incipient general paralysis, for he found this written on the head-card. It was pointed out to him that the resemblance was superficial, that the condition of the fundus and the absence of certain characteristic phenomena of general paralysis entirely negatived his diagnosis, and that the case was undoubtedly one of tumour, possibly syphilitic, situated somewhere about the base.

Dr. Mott thought, if a patient should come again under his care with such symptoms, he would be inclined after excluding syphilis to diagnose tumour of the 3rd ventricle and intracranial pressure from distension of the lateral ventricles, the condition which was found in these cases. These tumours were non-malignant, and produced symptoms by pressure upon the choroidal veins and the vein of the basal ganglia opening into the veins of Galen. Exudation into the lateral ventricles ensued, and drowsiness, deep sleep, and coma, with and without convulsions, the result of circulatory disturbances induced by the internal hydrocephalus.

As soon as the fluid drained off the circulatory disturbance passed off, and the patient was for a time partially restored. Such a syndroma as had been related in these cases appeared to him to indicate drainage of the lateral ventricles, and he would recommend the method of Watson Cheyne.

The full account of the cases and their pathological points of interest would be published shortly in the "Archives of the Pathological Laboratory of the London County Asylums."

VII.—DOUGLAS STANLEY, M.D.EDIN.

THE following are the notes of a case of *thalamic tumour* under the author's care at the Queen's Hospital, Birmingham. The patient was a clerk, aged 34, who had been addicted to alcohol, but without syphilitic history. For six weeks previous to admission he had been in low spirits from having lost his situation, and had in consequence been drinking freely. He woke one morning with a feeling of nausea, and vomited two or three times. During the afternoon he became drowsy and stupid, and the vomiting was repeated. He was admitted on the third day from the commencement of his symptoms. He was dull and stupid and could not answer questions readily, but he was able to indicate that he suffered from severe headache both "back and front." He answered questions in a slow hesitating manner. Physical examination did not reveal any sensory alteration. There was some slight paresis in the right arm and leg (dynamometer, left 60, right 35). There was marked difficulty in controlling volitional movements of both arm and leg on the right side. When asked to touch the tip of his nose with his eyes shut, he made several attempts before finally succeeding. He was barely able to feed himself, chiefly on account of a coarse, irregular tremor of the right arm. His gait was unsteady and he could barely walk without support, and there was reeling to the right side (screwing in). On attempting to stand with his feet together, he tended to fall to the right side. The organic reflexes seemed unaffected. The plantar reflexes were increased, and the cremasteric, epigastric and abdominal were present. The knee-jerk was absent on each side. There was well marked double optic neuritis.

After admission, the patient improved somewhat as regards his mental condition and the vomiting, but after a short time these symptoms returned, and the right-sided paresis increased. Occasionally convergent strabismus appeared on the right side, but the diplopia was unascertainable. There was also slight paresis of the right side of the face, especially of the muscles about the angle of the mouth, but there was no difficulty in closing the eyes. Incontinence of urine appeared later on, and it was found that he called objects by wrong names. It was also noticed that he had an occasional internal squint with the left eye which varied in degree, so that there was alternating converging strabismus, and it was often difficult to say which external rectus was most affected. The right angle of the mouth remained blurred. About fourteen days after admission, there was protrusion of the tongue to the left side and considerable increase of paresis of the right arm and leg amounting finally to almost complete paralysis. About a month after admission, the temperature rose suddenly to 104·6°, breathing became stertorous, pulse 116, the patient became cyanosed and died. On *post-mortem* examination, a large tumour was found occupying the whole of the left optic thalamus, causing compression of the internal capsule and displacement outwards of the lenticular nucleus, with projection into the lateral ventricle so as to cause some slight pressure on the opposite thalamus. The tumour was of different consistence, being much firmer posteriorly than anteriorly. In its substance was a considerable degree of hæmorrhage. Its nature was a glio-sarcoma.

VIII.—W. A. TURNER, M.D., F.R.C.P.

DR. ALDREN TURNER proposed to confine his remarks to a few cases of tumour occupying the posterior cranial fossa.

Corpora quadrigemina.—In the first place, there were two cases of tumour involving this region, one of which had been already recorded by Dr. Guthrie and himself in the *Lancet*, 1895, p. 273.

In both cases the onset of the disease was characterised by *headache*, which varied in position; while later on a *staggering gait* combined with *diplopia* were the chief symptoms, both coming on about the same time. In one case the inclination was invariably backwards, so that the patient was unable to stand or even sit up in bed without support from behind.

The *ocular* troubles consisted in paresis of the third and sixth nerves in one case, and defect in the upward and downward movements of the globes in the other. In both there was double optic neuritis; but no hemiopia or visual impairment, other than could be explained by the state of the fundus oculi. In one the knee-jerks were lost, in the other they were less active than natural.

Both these cases confirm the generally accepted symptomatology of quadrigeminal tumours, viz.: the association of ataxy of a cerebellar character with impairment in the movements of the eyeglobes, and without visual defect other than may be caused by the optic neuritis, which usually co-exists.

In some cases of tumour in this locality tremors of an intentional character have been observed; these are probably due to extension of the growth forwards into the tegmentum cruris so as to involve the superior cerebellar peduncle.

Cerebellum.—There were two cases of tumour occupying the posterior cranial fossa, both of which pressed upon the *left* middle cerebellar peduncle, and sprang apparently from the neighbourhood of the internal auditory meatus and posterior surface of the petrous temporal bone.

In both *deafness* of the left ear was an early and progressive symptom, and arose from pressure upon and eventual destruction of the eighth nerve upon the side of the lesion. In both the knee-jerks were retained, but that on the side of the lesion was more readily obtained and more active than that on the opposite side. In one the headache was mainly occipital, and in the other there was a tendency to fall to the side away from the tumour.

These cases do not add to our knowledge of the symptomatology of cerebellar tumour except in so far as unilateral deafness of nervous nature and one-sided exaggeration of the knee-jerk point to the side on which the tumour lies.

Vermis Cerebelli.—There was one case of tuberculous tumour involving the medulla of the superior vermiform process, more especially its anterior part. In this the headache was throughout frontal in situation; the knee-jerks were equal and active, and the patient was quite unable to stand or walk owing to the retraction of the head and marked inclination backwards of the body. The legs also were maintained in a state of rigid extension which was sometimes more marked in the right, at other times in the left leg.

Though in many respects the symptoms of tumour of the anterior vermiform process resemble those of tumour of the quadrigeminal region, the absence of defective ocular movements, or of ocular palsies, in the former is usually a reliable means of distinguishing them.

From these cases it is also apparent that the state of the knee-jerks in tumour of the posterior cranial fossa is an uncertain quantity. In some there is no jerk, in others the jerks are exaggerated, while in others one jerk is natural and the other is increased in force. An explanation of this variation may be found in the many altered physiological conditions which a tumour in this region is capable of producing; but more especially through the influence which a new growth has upon the general intra-cranial pressure by interference with the circulation in the veins of Galen, and with the flow of cerebro-spinal fluid through the aqueductus Sylvii.

A REMARKABLE CASE OF APHASIA.

Acute and Complete Destruction by Embolic Softening of the Left Motor-Vocal Speech Centre (Broca's Convolution), in a Right-Handed Man: Transient Motor Aphasia, Marked Inability to Name Objects and Especially Persons, Considerable Agraphia and Slight Word-Blindness.

BY BYROM BRAMWELL, M.D., F.R.C.P.EDIN.; F.R.S.EDIN.

Physician to the Royal Infirmary, Edinburgh; Lecturer on the Principles and Practice of Medicine and on Clinical Medicine in the School of Medicine of the Royal Colleges, Edinburgh; etc., etc.

It is, I think, no exaggeration to say that the case which I am about to relate is one of the most remarkable cases of aphasia which have ever been recorded, and that it is of very great importance both from a physiological and a pathological point of view. The details are as follows:—

A. B., aged 70, was seen with Dr. Menzies on March 4, 1898, at 11 a.m.

Previous History.

The patient has been a strong robust man, in the habit of attending business with great regularity. Some years ago, he suffered from symptoms suggestive of angina pectoris; at that time Dr. Menzies suspected the presence of fatty heart. After a few weeks, he completely recovered from this attack, and has been perfectly well ever since. Of late years, he has had a good deal of business worry.

History of the Present Illness.

His wife gave me in writing the following account of the commencement of the present attack:—

"On February 28, I left Mr. B. at his office at half-past four o'clock, in good health and spirits. He came home by the train which arrives at —— Station at a few minutes after six o'clock. I understand that as he was coming up the steps leading from the station his umbrella dropped from his right hand, but he was able to pick it up and to gain the landing. He then entered an omnibus which was waiting and which passes our door. On alighting from the omnibus, he opened the door with his key and came into the dining room. He then took off his boots and put on his slippers, but did not speak a word. I was surprised at this. I asked him what was the matter, but he only shook his head. I, at first, thought that he must have had some heavy business loss, but soon saw that he was ill. I became much alarmed and gave him a little whisky-and-water; this seemed to give him some relief. We then had tea; he was not able to eat anything, but drank a little tea. He then left the room and went upstairs to the bath-room; I followed shortly afterwards and found him smoking. I asked him if he felt any better, and he said, 'Yes.' I waited with him until he came down-stairs. After coming down to the dining room he took up the newspaper and tried to read, but he soon tired of it. I then asked him to lie down on the sofa and rest, but he would not do so; *he said he would take a game at bezique;* we had a game, but he made mistakes of which I took no notice. I thought he had had a shock of some kind, but he rather resented the idea of being ill and would not allow me to get a doctor. During the evening he spoke very little, but he said some words—for example, he said that he 'would have a game at bezique.'

"*Next morning* (*March* 1), he would not be persuaded to lie in bed, but rose as usual at six o'clock. His speech was better than on the previous night. We had breakfast at seven, and I prepared myself without his knowledge to accompany him to town. He was not very well pleased when he saw me dressed, and asked me where I was going; I said I wished to go with him. I went to the station, but he forbade me to come further, and I saw that if I persisted it would only make him angry. I was, therefore, forced very unwillingly to go home again. In the forenoon, I went to his office and saw him; he looked very well and said he felt better. I, however, without his knowledge, went along and saw Dr. Menzies, who advised me to persuade Mr. B. to go home as soon as possible, and promised to come out and see him. I expected to have some difficulty in getting him to go home with me, but he went quite willingly."

Dr. Menzies, who saw the patient on the afternoon of March 1 (the day after the onset), gave me the following account of his condition :—

"Mr. B. was in the drawing-room. When I went into the room he rose up and shook hands with me, and said, 'How do you do? How do you do?' He then spoke about the weather. At first, while he was speaking about ordinary things, I noticed nothing wrong; there was no hesitation and no thickness of speech. After some conversation I asked him, 'Do you know me?' and he said, 'Yes, yes, perfectly, of course.' I then said, 'Well, what is my name?' but he could not tell me. He understood everything that was said to him, and correctly answered the questions which I put to him. He seemed at a loss for some words, but the chief defect in speech, which I was able to make out, was that he could not name persons and objects. I noticed no paralysis of the face, but I thought he dragged the right toe slightly in walking. The pulse was slow —50 per minute. I then tested his writing and found that he could write very little. I advised that he should keep indoors and not go to business the next day. He did not, however, accept this advice."

The next day (*March* 2), he insisted upon going to business. While at business he was unable to write; he spoiled three cheques in trying to sign them.

On Thursday, March 3, he kept his bed. He had been annoyed about the mistakes he made in drawing the cheques the previous day, and realised that he was ill and unable to do his usual work. The temperature was 100°. He was speaking better than the day before, saying more words. I tested his power of writing and found he was able to sign his name fairly well.

PRESENT CONDITION (FRIDAY, MARCH 4, 1898).

When I saw him at 11 a.m. he was in bed (he had been kept in bed for the purpose of my examination). He was a fresh, robust-looking man, and looked younger than his age.

I entered into conversation with him; he seemed to understand everything that was said to him, spoke quite rationally and properly, and did not seem to be at a loss for words. I could detect no defect in speech until I came to ask him to name persons and objects. Dr. Menzies had previously told me that he was unable to name persons or objects. When I asked him what his own name was he could not say it;

he could not name Dr. Menzies or his own wife. When I asked him, "What is your wife's name?" he said, "Sugar and spice" four times over. (His wife was not in the room when the examination was made.) I then showed him various objects—a knife, a button-hook, a tuning-fork, a paper-knife—but he was unable to name any of them. A key he said was "a lock" (paraphasia); this was the only attempt which he made to name any object shown to him. He was able to repeat his own name and the names of objects which he was unable to name spontaneously.

There appeared to be a slight degree of word-blindness.

He could read aloud fairly well. He was asked to read aloud the following heading to a prospectus which was lying on the table:—*The Edison-Bell Consolidated Phonographic Company, Limited.* He read it correctly except the word *Phonographic*, which he read as "Photographic." He was then asked to read the following sentence:—*List of some of the Users of the Commercial Phonograph—Continued.* The word *Users* he read as "Liners," the word *Commercial* as "Farewell" and the word *Phonograph* as "Photograph." He read the word *Dog*, which I had written down, correctly.

His writing was then tested. He was first asked to sign his name, and did so correctly, the signature being not at all bad. He was then asked to write his wife's name; he again wrote his own name. He was next asked to write Dr. Menzies' name; he again wrote his own name. Asked to write the letter *o*; wrote *o* followed by two words which looked like *of zinc*. The numbers 20 and 100 were written in the same way—20 correctly, but it was followed by *of zinc* or *of June*. He was able to read his own name, which I had written. He was then asked to write his wife's name, and he wrote "Dear Sir," "Dear Surgises," several times over—in fact, he wrote this or his own name for almost everything he was asked to write. When asked to write the words "one hundred," he did so correctly. Asked to write a thousand, wrote "one forager." He read several words which he could not write. Was again asked to write the number 20; wrote "twevry flory" followed by "20" in figures. When asked to write down the individual letters *a*, *s*, *o*, he failed to do so.

SUMMARY OF THE SPEECH DEFECTS.

The chief defect was inability to name persons and objects. There was also a considerable degree of agraphia and paragraphia; a certain, though slight, degree of word-blindness, and some difficulty in reading aloud, which was perhaps a motor defect. Except for naming objects and persons (if this was a motor defect), there appeared to be no motor-vocal aphasia. There was no word-deafness.

The patient was a right-handed man and had always been so. After the *post-mortem* examination I again made particular enquiries as to this point. His wife assured me that her husband had always been right-handed and had never used his left hand more than any ordinary right-handed person is in the habit of doing. His sister stated that as a child the patient was right-handed and that none of the family (father, mother, two brothers and two sisters) were left-handed.

Except the aphasic defects described above and a slight degree of tremor of the right hand, apparently due to commencing paralysis agitans, there was absolutely no indication of any nervous lesion. I could detect no paralysis either in the face, tongue, arm, or leg. The knee-jerks were equal and normal. There seemed to be no disturbance of sensation; in particular there was no hemianopsia.

His wife stated that for the first two or three days after the attack the patient had fed himself with his left hand. She further said that he had been shaky in his right hand for some considerable time, apparently the result of slight paralysis agitans; she did not think he was more shaky since the attack came on than he was before.

The heart seemed somewhat dilated, the aortic second sound accentuated, the first sound in the mitral area impure. The urine was free from albumen.

Diagnosis.

The diagnosis was embolism of one of the branches of the left middle cerebral artery, with softening in some part of the speech area. The symptoms seemed to me to suggest that the lesion was situated in the neighbourhood of the visual speech centre, and that the impulses passing from this centre (and perhaps also from the auditory speech centre) to the motor speech centre (Broca's convolution) and to the motor centre for writing were probably, in part at least, interrupted.

There was no evidence of any lesion in the auditory speech centre.

The fact that motor speech was not affected, except for naming objects and persons (if that was a motor defect) seemed to show that the motor-vocal speech centre (Broca's convolution) was not directly affected by the lesion.

The patient's inability to name persons and objects was the most striking speech defect, and at the time of the consultation Dr. Menzies said: "If there is a naming centre, that is the seat of the lesion in this case." The patient was not only unable to spontaneously name persons (such as his wife—who was not present in the room when he was asked to name her), but was unable to name objects which were shown to him.

Subsequent Progress of the Case.

I did not see the patient again during life, but Dr. Menzies has kindly given me the following account of the subsequent progress of the case:—

"After our consultation the patient improved slowly, but the aphasic symptoms still persisted in some degree. The great difficulty was in recalling the names of persons. There was no other defect in motor speech; for example, I would go in to see him and he would talk quite well, say 'How are you to-day, Doctor? It is a cold morning.' I would ask him, 'How do you do?' He would say, 'Quite well, thank you,' etc. Then I would ask him at what hour he got up this morning, and he would tell me he got up at seven o'clock. I would then ask him what he had for breakfast, and he would tell me correctly. He would talk in this way for about five minutes, and then I would ask him, 'Do you know me?' and he would say 'Yes, quite well, perfectly.' Then I would say, 'Well, what is my name?' and he could not name me. Then I would ask his wife's name, and he could not name her. He was annoyed that he could not do so. Except during the earlier stages of the case, I did not notice any other defect of vocal speech except his inability to name persons and objects.

"He continued to have considerable difficulty in writing for some time after our consultation. He usually could write his own name, but tended to introduce more letters than were necessary, and the same with numbers.

"*On March 9* he wrote my name correctly when asked to write 'Dr. Menzies;' after an interval, when asked to write it a second time, without the name being mentioned, he wrote 'Dr. Magnession.' He was then asked to write his own name and did so correctly; asked to write it a second time, wrote 'Dr Dr do do'; asked to write it a third time, wrote 'Dr Dr do do' (meant for ditto). Was asked to write a hundred, wrote it correctly, then added, '£100-0-0' in figures and his own name; 'one hundred £100 0 0' and his own name. Wrote the letters A, C, D, Z, N correctly when asked to do so. Wrote 'One hundred pounds,' his own name and address, when asked to do so. When asked to write his wife's name (the name not being mentioned to him) wrote 'Mrs. Bagginny.' When asked to write Dr. Menzies' name, wrote 'Mr. Dr. Menzies.'

"He knew my name, and sometimes if I asked him to write my name, Dr. Menzies, he would write it; and if I gave him what he had written before—perhaps on some previous occasion he had written my name—he could read it, but only by spelling it out, letter by letter, and then he knew that that was my name (persistence of some word-blindness). He never could say spontaneously my name or his wife's name. Sometimes he could name objects which were shown to him; for example, on one occasion I picked up a cup from the table in the drawing-room and asked him what it was, and he named it correctly. Names of persons and objects which he could not himself spontaneously utter he could repeat. For example, on one occasion on which he could not mention his own name, after I named him he said, 'Yes, A—— B——, of course, A—— B——.' He could repeat everything (names, etc.), which he was asked to repeat.

"His power of reading aloud gradually improved. During the course of the illness he made his will; he was quite capable of making it. He asked me to arrange this for him, and I advised him to get a lawyer to draw it up for him. When the lawyer visited him for the purpose of making his will, he spoke quite well and explained what he wanted. The lawyer then read over the will, explained its general provisions and asked him if it was what he intended to do, and he said, 'Yes, perfectly.' He made no alterations or additions. The will was a very simple one; he left everything to his wife.

"A peculiar thing was that in his previous illnesses it was difficult to get him to remain at home or to lay up; but in this illness, after the first few days, he seemed to be indifferent about his business; he did not even care to go out, and rarely made an attempt to do so.

"*On Friday, April* 8, he went to business. He had been down to his place of business, but had not done much, on several previous occasions. (On this day his wife says that he was better than he had been since the attack commenced.) He remained at his business the whole day, visited his two business places in Edinburgh and Leith, looked through the books, took an intelligent interest in what was going on, gave instructions, etc. His foreman did not notice that there was anything the matter with him. He made no mistakes. He drew and signed several cheques quite correctly. I have seen the counterfoils of these cheques, and I find that he had himself written the names of two or three firms and had spelt these names correctly. The cheques were drawn by his own hand.

"He never wrote any letters during the course of his illness. He read very little, but he never had been a great reader. He never complained of his sight.

"*On April* 9 he complained of pain in the region of the stomach. I saw him the next day (April 10). He did not look ill, but there was considerable tenderness over the abdomen. The pulse was slightly quickened—about 80 per minute—but there was no temperature. Up to this date, from the onset of the aphasic attack, the pulse had been slow (45 to 50 per minute).

"*On April* 11 the temperature went up to 100°, and the pulse to about 96. He complained of intense pain over the abdomen and great tenderness on pressure, and he vomited frequently. The abdomen was moderately distended. Peritonitis was obviously present. During the attack the bowels were more than once freely opened by medicine. There was no intestinal obstruction and no apparent cause for the peritonitis.

"*On April* 12 *and* 13 he remained much *in statu quo.*

"*On April* 14 he vomited a small quantity of blood.

"*On April* 15 he died. He remained mentally quite clear up to the end. On the day of his death he drew and signed a cheque quite correctly; the writing was good."

POST-MORTEM EXAMINATION.

The *post-mortem* examination was made on April 16, 1898, at 3.30 p.m., by myself, Dr. Menzies and Dr. Steven being present.

The body was well nourished. The skull cap was thin, the dura mater very firmly adherent along the vertex. Several marked depressions in the bone were present, the result of the pressure of Pacchionian glands on the vertex, where the dura was adherent.

The convolutions of the *brain* were slightly atrophied. There was a marked depression in the position of Broca's convolution and the lower end of the left ascending frontal convolution. The exterior of the brain was otherwise normal. The brain was at once placed in formalin, and after it had become sufficiently

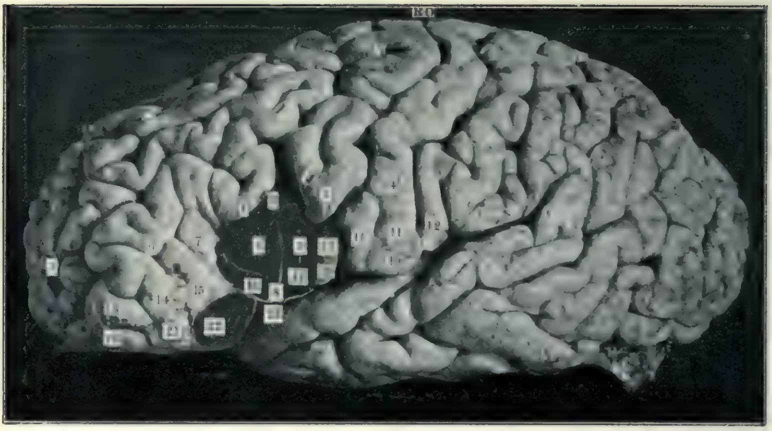

Fig. 1.

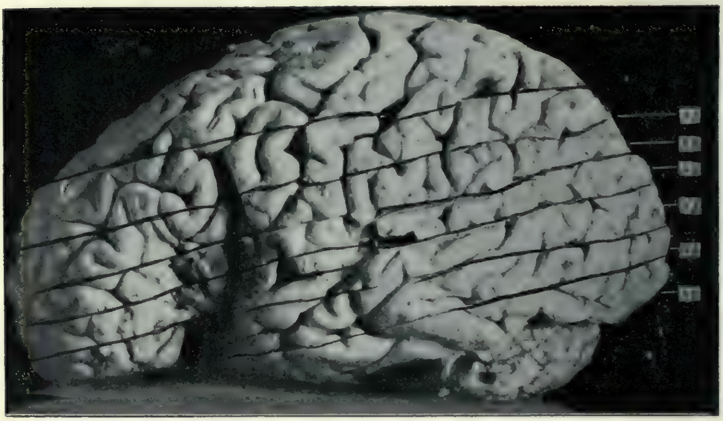

Fig. 2.

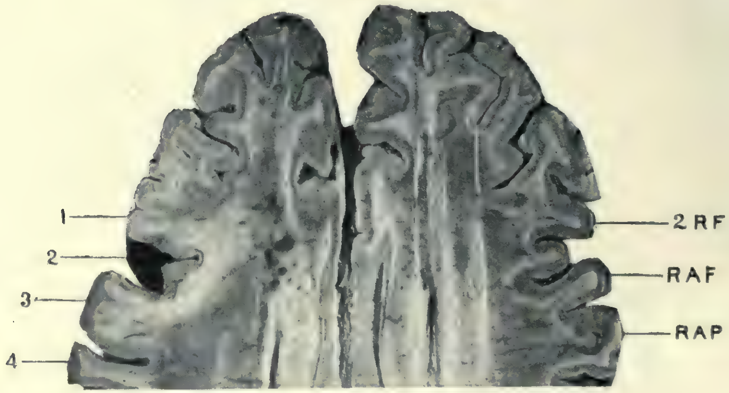

Fig. 3.

hardened was cut into a series of horizontal sections. A more minute description of these sections is given below.

On opening the abdomen several coils of the small intestine were found to be black and gangrenous, and there was general peritonitis. There was no constriction and no hernia, either externally or internally. The portion of mesentery going to the affected part of the small intestine was markedly swollen and œdematous. The gangrene of the intestine was obviously due to blocking of the mesenteric artery. The abdominal aorta was somewhat atheromatous.

The *heart* was moderately dilated, the valves healthy, the muscular substance markedly fatty and degenerated. The *thoracic aorta* was atheromatous. The *kidneys* were slightly enlarged and somewhat granular. (The urine was tested on several occasions during the course of the patient's illness, and was always free from albumen.) A few recent small petechial hæmorrhages were scattered over the mucous membrane of the *stomach.*

No other lesions were present in the body.

Subsequent Examination of the Brain.

After the brain was sufficiently hardened in formalin (10 per cent. solution), the left and right hemispheres were photographed with the membranes *in situ.*

The photograph of the left hemisphere shows a marked depression (shrinking due to softening) in the position of the posterior end of the lower (3rd) left frontal convolution (Broca's convolution).

The membranes were then carefully removed, and the left and right hemispheres were again photographed.

The depression (due to softening and atrophic shrinking) in the position of the posterior end of the lower (3rd) left frontal convolution was still more apparent.

The extent of the softening is exactly represented in fig. 1.

A small portion, the uppermost portion, of Broca's convolution (on which the figure 2 in fig. 1 is placed) appeared, at first sight, to have escaped; but although it was not shrivelled and completely destroyed, it was markedly affected (softened). This is not brought out in the photograph, but is distinctly seen in the brain itself.

The part of the lower (3rd) left frontal convolution on which the figure 22 in fig. 1 is placed, and a part of the convolution on which the figure 7 is placed, were also markedly softened; this is well seen in the actual preparation and in the longitudinal (horizontal) sections reproduced in figs. 3, 4, 5 and 6, and in fig. 7.

When I first examined the specimen I was disposed to think that the lower end of the left ascending frontal convolution was affected (softened and atrophied), but subsequent examination showed that this was not the case. I may here state that I have had the advantage of Sir William Turner's opinion, and that he agrees with the description which I have given of the locality of the lesion and the arrangement of the convolutions in this case.

In this particular brain the lower end of the left ascending frontal convolution (on which the figures 10 and 18 in fig. 1 are placed) is relatively narrow (small), and the "foot" and ascending portion of Broca's convolution (on which the figures 9 and 17 are placed) is relatively broad (large). At first sight I was consequently disposed to think that the posterior end of the lower (3rd) left frontal convolution was the lower end of the left ascending frontal convolution.

The figures in fig. 1 and in figs. 3, 4, 5 and 6 point to identical parts of the convolutions. By this procedure the exact localisation of the softening and of the different convolutions in the longitudinal (horizontal) sections can be determined at a glance.

The brain was then cut into a series of longitudinal (horizontal) sections, and *the upper surface* of each of these sections was then photographed.

Finally, the sections were placed in accurate apposition, and the brain (left side) was again photographed (see fig. 2) so as to show the exact level of the various sections (1, 2, 3, 4, 5 and 6).

The *first section* (to which S. 1 in fig. 2 points) was absolutely normal, and has not therefore been reproduced.

The *second section* (see fig. 3) to which S. 2 in fig. 2 points, shows a small area of softening, which corresponds to the uppermost point of Broca's convolution, the point on which the figure 2 in fig. 1 is placed.

The *third section* see fig. 4) to which S. 3 in fig. 2 points, shows a marked area of softening involving the posterior end of the lower (3rd) left frontal convolution and the anterior end of the island of Reil.

The *fourth section* (see fig. 5) to which S. 4 in fig. 2 points, shows a still more extensive area of softening, which involves Broca's convolution, and the grey matter of the anterior end of the left island of Reil.

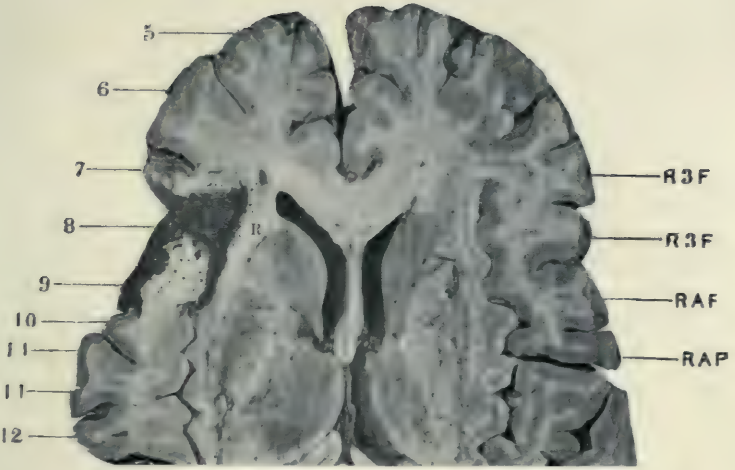

Fig. 4.

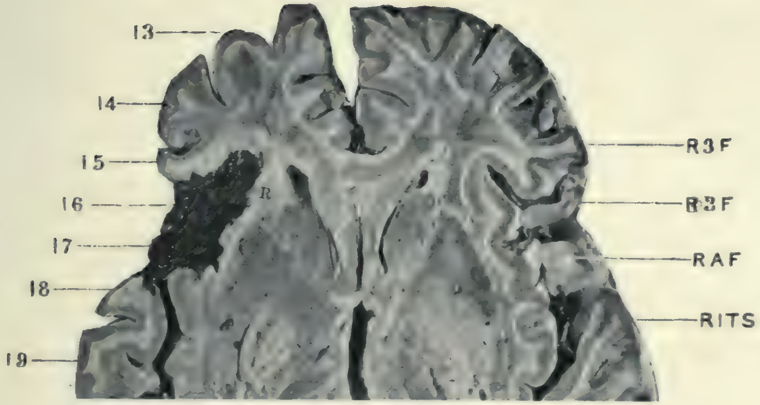

Fig. 5.

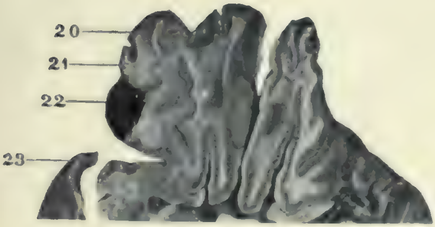

Fig. 6.

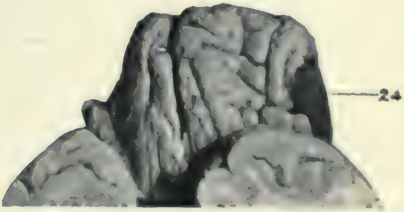

Fig. 7.

The *fifth section* (see fig. 6) to which S. 5 in fig. 2 points, shows a small area of softening corresponding to the orbital part of the lower (3rd) left frontal convolution.

It should be noted that this section was not quite horizontal, the right frontal lobe is cut through at a lower level than the left; hence the portion of the left frontal lobe which is seen in the section is considerably larger than the portion of the right frontal lobe seen in the section.

Finally, the inferior surface of the frontal lobes was photographed; in other words, section 5, fig. 2, was reversed and its under surface photographed.

The small part (anterior end of the left inferior frontal convolution) to which the figure 24 points and which is shaded black, was distinctly affected (yellowish-brown in colour and softened). This is not apparent in the photograph, but it is very apparent in the actual preparation.

I took the opportunity of showing both the sections themselves and the photographs to Professors Ferrier and Joseph Collins at the recent Edinburgh Meeting of the British Medical Association, and these authorities spontaneously remarked that the area of softening was much better seen (owing to the difference in colour) in the actual sections than in the photographs, and was more extensive than one would have supposed from an examination of the photographs.

REMARKS.

The point in this case which I wish to specially emphasise, for it seems to me to be of supreme interest and importance, is that in this patient, who was a right-handed man and in no sense more left-handed than the great majority of right-handed persons, acute and complete destruction of the left motor-vocal speech centre (Broca's convolution) and of the anterior end of the left island of Reil merely produced a very temporary motor aphasia, and did not produce, as one would have expected it to produce, complete and persisting motor-vocal aphasia.[1]

All authorities on aphasia are agreed:—(1) that in right-handed persons the motor-vocal speech centre is situated in the posterior end of the lower (third) left frontal convolution (Broca's convolution); in other words, that Broca's convolution is "*the way out*" for spoken (vocal) speech; and (2) that complete destruction of the left motor speech centre (Broca's convolution), if acutely produced in a right-handed person, should produce complete and persistent (though not, of course, necessarily permanent) motor aphasia, or, as I term it, motor-vocal aphasia.

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[1] I use the term *persisting* in contradistinction to *temporary*, but it must be remembered that the term "persisting" is not synonymous with *permanent*, for in some cases in which aphasic symptoms persist for some time they are not permanent.

Up to the present time almost all authorities on aphasia have emphatically maintained:—(1) that, in right-handed persons, the speech centres are located in the left hemisphere of the brain; and (2) that the corresponding cortical centres in the right hemisphere are not concerned with speech. Though almost every one will, I presume, allow that the corresponding centres in the right hemisphere can, in some cases at all events (more especially in young subjects), be trained to take up and carry on the function of the speech centres in the left hemisphere, when the speech centres in the left hemisphere are destroyed; and that under such circumstances the way out for spoken speech is through the posterior end of the third *right* frontal convolution.

Consequently, according to the usually accepted view, complete destruction of Broca's convolution, if acutely produced in a right-handed person, will *necessarily* (*i.e.*, always) cause complete and persisting motor-vocal aphasia; and the aphasic defects produced in this way can only be recovered from (and in the adult this recovery is usually very imperfect) by a slow and gradual process of education and training of the corresponding (motor-vocal speech) centre in the right hemisphere of the brain.

The case which I have related above seems to me to show that these statements do not invariably hold good; in other words, that exceptions to the usually accepted theory do occasionally, though, of course, I admit *very rarely*, occur. It proves, in my opinion:—(1) That acute and complete destruction of Broca's convolution, in a right-handed person, does not necessarily produce complete and persisting motor-vocal aphasia, but that, in exceptional cases, such destruction may merely cause a very temporary and slight degree of motor-vocal aphasia.

(2) That in right-handed persons "the way out" for speech is not necessarily and invariably through Broca's convolution (the posterior end of the lower *left* frontal convolution).

It seems obvious that in this right-handed man, after the occurrence of the lesion, motor-speech impulses did not pass out through Broca's convolution, for almost immediately after the occurrence of the lesion, *i.e.*, almost immediately after Broca's convolution was completely destroyed, motor-speech impulses continued to pass out in what was to all intents and purposes a normal manner.

The question therefore occurs, through what part of the brain did they pass out?

There can only, I think, be one satisfactory answer to this question, viz., through the posterior end of the lower (3rd) frontal convolution *on the right side of the brain*, unless the terms "Broca's convolution" and the "motor-vocal speech centre" are allowed to include the inferior end of the left ascending frontal convolution, which was not implicated by the lesion in this case.[1]

The only theory which seems to me to afford a satisfactory explanation of the facts of this exceptional case is that, in this patient, the cortical centre in the right hemisphere corresponding to Broca's convolution, or the right motor-vocal speech centre, as I term it, was more highly educated than it is in the great majority of right-handed people—in other words, that, in this particular patient, the motor-vocal speech centre in the right hemisphere was sufficiently educated and active to *immediately* take up and carry on the function of the left motor-vocal speech centre when that centre was *acutely* destroyed.

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[1] Recent observations would seem to show that the lower end of the ascending frontal convolution (which is separated from the posterior end of the 3rd left frontal convolution by the inferior end of the præ-central sulcus) is the ordinary psycho-motor centre for the lower part of the face, tongue and larynx, and does not form a part of the motor-speech centre properly so called. Further, it is usually supposed that the motor-speech centre does not include the whole of Broca's convolution, but is limited to that portion of the 3rd left frontal convolution which is bounded behind by the præ-central sulcus and in front by the ascending limit of the fissure of Sylvius (*i.e.*, the "foot" and ascending portions of Broca's convolution). According to this view, the portion of Broca's convolution (the "cap") which is situated in front of the ascending limit of the fissure of Sylvius does not form part of the motor-speech centre.

For several years past I have been in the habit of teaching:—(1) that the commonly-accepted view that the left hemisphere is alone concerned with speech in right-handed persons (and the right hemisphere in left-handed persons) is erroneous; (2) that, in right-handed persons, the cortical centres in the right hemisphere (corresponding to the speech centres in the left hemisphere) must be possessed of some sort of speech function (and the reverse in left-handed persons); (3) that, in right-handed persons, the relative activity of the different speech centres (the auditory speech centre, the visual speech centre and the motor-vocal speech centre) in the right hemisphere is probably different in the same individual, the right auditory speech centre, for example, being, I think, in most individuals, probably more highly educated and more active than the right motor-vocal speech centre; and (4) that, in right-handed persons, the relative activity of the speech centres on the left and right sides of the brain, or perhaps it would be more correct to say, the relative degree of activity which the different speech centres in the right hemisphere of the brain possess (for, so far as our present information enables us to judge, in right-handed persons the speech centres in the left hemisphere are always the "active" or "driving" centres), probably varies considerably in different individuals.[1]

The experience of all observers goes to show that the motor-vocal speech centre in the right hemisphere (though, in my opinion, it probably possesses in every right-handed individual some sort of active speech function, which, I presume, is carried on in conjunction with, *and in subordination to*, the function of the active or "driving" motor-vocal speech centre in the left hemisphere) is in the great majority of right-handed persons not sufficiently educated and active to immediately take up and carry on (*i.e.*, to independently carry on) the motor speech-function when the active or driving centre (Broca's convolution) in the left hemisphere is acutely and completely destroyed.

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[1] In my Lectures on "Aphasia" (published in the *Edinburgh Medical Journal*, vol. ii., 1897) I have thrown out the suggestion that in some right-handed persons whose ancestors were left-handed the active or driving speech-centres may *possibly* in *rare instances* be situated in the right hemisphere; but while this is, I think, theoretically probable, no case in which it actually occurred has, so far as I know, been recorded.

But that, in some persons, more especially in young subjects, the motor-vocal speech centre in the right hemisphere may, in the course of a short time, become so perfectly trained and educated as to be able to actively carry on the speech function without any obvious defect, after Broca's convolution (the active or driving motor-vocal speech centre) has been acutely and completely destroyed, is *conclusively proved* by Barlow's well-known case.

Now, the present case seems to me to show that, in very rare and quite exceptional cases, the right motor-vocal speech centre in right-handed adults is sufficiently educated and active to *immediately* take up and carry on (*i.e.*, to independently carry on) the speech function (the emission of motor speech-impulses) when Broca's convolution is acutely and completely destroyed.

And if this be allowed, it is reasonable, I hold, to maintain (as I have long argued) that the degree of functional activity and educational endowment which the right motor-vocal speech centre (and indeed all the speech centres) possesses in different right-handed persons is probably variable. I presume :—

(1) That in the vast majority of right-handed persons the functional activity of the right motor-speech centre is probably slight—so slight as to be insufficient to enable it to *immediately* take up and carry on (*i.e.*, to independently carry on) the motor-vocal speech function in any obvious or in any considerable degree when the driving motor-vocal speech centre (Broca's convolution) is destroyed.

(2) That in very rare instances, of which the present case is, I suggest, an illustration, its functional activity and educational endowment are so perfect that the speech function can be *immediately* taken up and, practically speaking, almost perfectly carried on when the left motor-vocal speech centre is acutely and completely destroyed.

And (3) that between these two extremes there are probably all degrees of difference; though, as I have already remarked, the experience of all observers goes to show that the cases in which the right motor-vocal speech centre is sufficiently educated and active to carry on the speech function under such circumstances (*immediately* after acute and complete destruction of Broca's convolution), with any degree of completeness, are altogether exceptional.

I maintain, then, that this case seems to me to corroborate and strongly support the view which I have for a long time advocated, that the speech centres and speech functions are bilaterally represented in the brain—the view which supposes that, in right-handed persons, the cortical centres in the right hemisphere which correspond to the speech centres in the left hemisphere must be possessed of some sort of speech function. Though, as I have already stated, I fully admit that, in the great majority of right-handed persons, the left hemisphere is, so far as the speech functions are concerned, the active or "driving" side; and that the degree of educational endowment and functional activity of the speech centres in the right hemisphere are usually quite insufficient to enable the speech function to be actively carried on when (immediately after) the speech centres in the left or active hemisphere are acutely and completely destroyed.

This view I prominently advocated in a series of papers and lectures which were published in the *Lancet*[1] in 1897 (March 20, *et seq.*); and in the *Edinburgh Medical Journal*, vol. ii., 1897, I have discussed the question in considerable detail.

ADDITIONAL POINTS OF INTEREST.

In addition to the point which has just been emphasised, the case presents several other points of considerable interest. Some of them are as follows:—

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[1] The papers in the *Lancet* (which appeared simultaneously with Dr. Charlton Bastian's lectures, in which a similar view as to the bilateral representation of the speech centres was advocated) had been in the hands of the Editor of the *Lancet* for six months before they appeared.

(1) *Absence of Paralysis of the Face, Tongue, &c.*

So far as I could detect, there was no paralysis of the face, lips, or tongue on the opposite (right) side five days after the occurrence of the lesion; and Dr. Menzies assures me that, when he saw the patient twenty-four hours after the onset of the attack, he did not observe any paralysis in these parts.

It seems certain, however, that the occurrence of the lesion was followed by some (temporary) motor weakness on the opposite (right) side of the body. This is shown by the following facts:—At the moment when, it is reasonable to suppose, the embolism occurred, the patient dropped his umbrella which he was carrying in his right hand; the next day Dr. Menzies thought that he dragged the right toe slightly; and for some days after the onset of the attack he did not feed himself with the right hand, as he had previously been in the habit of doing.

And since the onset of the lesion was attended with some (temporary) loss of motor power in the right arm, and apparently also in the right leg, it is reasonable to suppose that the onset of the lesion was probably attended with some motor weakness (paralysis, or paresis, as some writers would term it) in the muscles of the face, lips, tongue, &c., on the opposite (right) side of the body. (The temporary paralysis in the right arm, in (?) the right leg, and in (?) the right side of the face, tongue, &c., if there was temporary paralysis of the face, tongue, &c., was obviously due either to shock and inhibition or to temporary vascular changes produced by the lesion.)

But if such paralysis existed, it must have been very slight and it must have rapidly passed off. The very slight and very temporary character of this facial paralysis (if it did occur) is quite satisfactorily explained by the fact that the lower end of the left ascending frontal convolution (the ordinary psycho-motor centre for the lower part of the face, the lips, tongue and larynx) was not affected. That in man the lower end of the ascending frontal convolution is the ordinary psycho-motor centre for the lower face, lips, tongue and larynx, and that it does not form part of the motor-vocal speech centre seems shown by a case recorded by Elder in which the lower end of the left ascending frontal convolution was acutely destroyed with the result that there was paralysis of the lower facial muscles, tongue, &c., *but no motor aphasia*, Broca's convolution not being implicated by the lesion. Further, it must be remembered that many of the facial movements are bilaterally represented in the cerebral cortex and can be put into action from either hemisphere.

(2) *The Alterations in the Condition (functional activity) of the Visual Speech Centre, of the Motor Writing Centre, and perhaps of the Auditory Speech Centre, which were present in this case.*

The lesion, as we have seen, was strictly limited to the motor-vocal speech centre (Broca's convolution) and to the anterior end of the left island of Reil. But when I saw the patient (five days after the onset) and for some time afterwards, there was a certain (though slight) degree of word-blindness, considerable agraphia and paragraphia, and almost complete inability to name objects and persons. These symptoms were so marked and the motor aphasia was so slight (when I saw the patient there was, practically speaking, no motor aphasia, unless the inability to name persons and objects was a motor defect) that I was disposed to think that the lesion was situated in the neighbourhood of the visual speech centre, and that it probably more particularly implicated the fibres passing from the visual speech centre to the writing centre. I never for one moment suspected that the motor-vocal speech centre (Broca's convolution) was completely destroyed, as the autopsy subsequently demonstrated.

The case shows how difficult it is, in some cases, to predict the exact locality of aphasia-producing lesions; though I admit, of course, that this case is altogether an exceptional one, and I allow that, in the vast majority of cases in which one of the speech centres is acutely destroyed and in which the lesion is limited to that centre, the aphasic symptoms which result are sufficiently characteristic to enable one to localise the lesion with certainty and accuracy.

The slight degree of word-blindness which was present in this case cannot, so far as I see, be explained in any other way than by supposing that the destructive lesion of the motor-vocal speech centre produced temporary disturbance in the action of the visual speech centre. And if this is the correct explanation of the mode of production of the slight word-blindness, the case corroborates the view of those authorities (such as Déjérine, Mirallié and Joseph Collins) who maintain that the action of the different speech centres (auditory, visual and motor), which are comprised in what Freund terms the "*zone of language,*" is very intimately connected; and that a lesion in one centre (say the motor-vocal speech centre) is apt to produce (these authorities would, I suppose, say will necessarily produce) some disturbance in the action of the others (say the visual speech centre).

But here I would remark that it is impossible to argue on hard and fast lines, and to affirm that the same lesion will necessarily produce exactly the same symptoms or results in every case. It is of the utmost importance, I think, to recognise the fact that individual differences in the relative degree of the functional activity of the individual speech centres, *and of the closeness and intimacy of their functional activity and connections*, as it may be termed, occur in different cases; and that in one case in which this connection is close, a lesion (say of the motor-vocal speech centre) will produce considerable disturbance (say in the action of the visual speech centre)—as it seemed to do in this particular case; while in another case, in which the connection is less intimate, the same lesion will produce little or no disturbance. There is every reason, I think, to suppose that in some persons (those who are accustomed to read and write much) the action of the visual speech centre becomes so highly specialised and independent that the act of reading and writing can be largely (possibly entirely) carried on independently of the action of the motor-vocal speech centre; while in other persons (who are accustomed to read and write little) the action of the visual speech centre is much less highly specialised and independent of the action of the motor-vocal speech centre.

In this particular case, in which, I presume, the functional connection between the motor-vocal speech centre and the visual speech centre was intimate and close (and with reference to this point it may be of importance, as it certainly is of interest, to note that the patient had never been a great reader), destruction of the motor-vocal speech centre (Broca's convolution) produced a certain, though slight, degree of word-blindness and considerable agraphia.

But complete destruction of Broca's convolution does not always produce this result. In the *Lancet*, May 22, 1897, I have recorded a case in which complete and acute destruction of Broca's convolution produced complete motor-vocal aphasia, but no word-blindness and comparatively little agraphia.[1] In that case, I presume that the connection between the motor-vocal speech centre (Broca's convolution) and the visual speech centre (the left angular gyrus) had become, comparatively speaking, a loose one; in other words, in that case the action of the visual speech centre had become so differentiated, so highly specialised, that it was in the habit of acting much more independently of the motor-vocal speech centre than is often (? usually) the case.

And here I may note a very remarkable coincidence, viz., that both of these patients, who were not related in any way, had the same surname, which for obvious reasons I suppress, but which I may term "Brown."

Another point of interest between these two cases, in both of which the motor-vocal speech centre (Broca's convolution) was completely and acutely destroyed, was the condition of writing.

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[1] In this case there were two lesions. One, which occurred on November 21, 1892, was attended with complete motor-vocal aphasia, no word-blindness and comparatively little agraphia; the second, which occurred four months afterwards, was followed by some word-blindness and marked agraphia. The case in some respects forms a remarkable contrast to the one which I am at present recording.

In the present case, in which there was little or no motor-vocal aphasia (unless the inability to name objects and persons was a motor defect), but a certain though slight degree of word-blindness, there was considerable agraphia and paragraphia. In the former case, in which there was complete motor-vocal aphasia, there was no word-blindness and very little agraphia. The absence of any marked agraphia was forcibly illustrated by the fact that the patient, who was unable to answer questions in vocal speech, carried a notebook about with him for the purpose of answering questions and carrying on a conversation in writing.

The former case (recorded in the *Lancet*, May 22nd, 1897) seemed totally opposed to the view which the great majority of authorities on aphasia hold, that destruction of Broca's convolution (the motor-vocal speech centre) necessarily produces agraphia, and that, as Gairdner long ago argued, the degree of agraphia is usually proportionate to the degree of motor-vocal aphasia. It seems to support the view advocated by Bastian, which I had also independently arrived at, viz., that the nervous impulses which set the motor writing centre into action do not necessarily (and presumably, therefore, do not usually) pass through the motor-vocal speech centre in order to reach the writing centre, and that destruction of the motor-vocal speech centre does not necessarily (and presumably, therefore, does not usually) give rise to agraphia, although I, of course, admit the clinical fact that motor aphasia due to destruction of Broca's convolution is usually associated with agraphia and that the agraphia is usually (roughly speaking) proportionate in degree to the motor aphasia.

The present case seems, at first sight, to support the usually accepted theory. It shows that a lesion (complete destruction) of Broca's convolution (the motor-vocal speech centre) and of the anterior end of the left island of Reil which does not produce motor-vocal aphasia (unless the inability to name objects and persons was a motor defect) may produce considerable agraphia and paragraphia.

But while this must be admitted, it does not necessarily follow that the agraphia in this case was due to the interruption of nervous impulses passing through the motor-speech centre on their way to the writing centre; in other words, it does not necessarily prove that the nervous impulses which put the motor writing-centre into action *pass through* the motor-vocal speech centre.

The agraphia which was present in this case and which, it must be allowed, resulted either from the destructive lesion of Broca's convolution or from the destruction of the anterior end of the left island of Reil, for the lesion was sharply limited to these parts, may have been produced in other ways. Two theories may be advanced to account for the agraphia, viz.:—

(1) That the lesion of the anterior end of the island of Reil partially interrupted the conduction through the fibres which convey writing-producing impulses, as they pass from the visual speech centre to the motor centre for writing (which is, I think, probably situated in the ordinary psycho-motor centre for the movements of the right hand, but which Exner and some authorities think is perhaps situated in the posterior end of the second left frontal convolution).

(2) That the agraphia was an *indirect* result of the destruction of Broca's convolution, and was due to derangement of the action of the visual speech centre. In other words, the agraphia may have been due to the fact that destruction of Broca's convolution produced temporary functional disturbance in the action of the visual speech centre, and this functional disturbance in the action of the visual speech centre (in which, every one admits, writing impulses arise,[1] whether they afterwards pass through Broca's convolution or not, in order to reach the writing centre) was the cause of the agraphia. But, if this theory were correct, one would have expected the word-blindness to have been at least as great as the agraphia; and this was not the case.

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[1] In order not to complicate the argument too much, I omit any reference to the auditory speech centre in which the nervous impulses concerned in the production of writing probably originate.

I merely suggest this as a possible mode of production of the agraphia *in this particular case.* I do not mean, of course, to suggest that it is the usual mode of production of agraphia, in cases of motor aphasia due to a lesion involving the motor-vocal speech centre (Broca's convolution). I believe, with Bastian, that in most cases of motor aphasia in which there is agraphia, the agraphia is probably produced in one or other of the following ways :—(*a*) by the lesion which destroys Broca's convolution (and so produces motor-vocal aphasia), also implicating and destroying the motor writing centre (ordinary motor centre for the fingers and thumb, wrist, &c., of the right hand) ; or (*b*) destroying or interrupting the conduction through the fibres which conduct impulses to that centre (the motor writing centre) from the visual speech centre (*i.e.*, the fibres which *directly* connect the visual speech centre with the motor centre for writing).

The production of the agraphia in either of these ways is compatible with the theory that the nervous impulses which put the writing centre into action pass directly from the visual speech centre to the motor centre for writing ; in other words, that they do not necessarily (and presumably, therefore, do not usually) pass through the motor-vocal speech centre on their way from the visual speech centre to the motor centre for writing, as most authorities suppose.

That in this case, *after the occurrence of the lesion*, the nervous impulses which were concerned in the act of writing did not pass through the left motor-vocal speech centre (Broca's convolution) in order to reach the writing centre is conclusively proved by the fact that the motor-vocal speech centre (Broca's convolution) was completely destroyed.

But it is, of course, quite possible that such impulses may have passed to the motor centre for writing through the *right* motor-vocal speech centre, (which I maintain took up and carried on the motor-speech function) after Broca's convolution was destroyed.[1]

[1] I should here note that the patient's power of writing with the left hand was unfortunately not tested.

(3) *The Inability to name Persons and Objects.*

One of the most interesting points in the case (though it is altogether subordinate in importance to the central fact of the case, viz., that acute and complete destruction of the motor-vocal speech centre—Broca's convolution—merely produced a very temporary and slight degree of motor-vocal aphasia) is the fact that the patient was unable to name objects and persons. For some days after the onset of the attack he could not name himself, and throughout the subsequent stages of the case he was unable to name his wife or his doctor. This inability to name persons and objects was such a striking feature of the case that at the time of our consultation Dr. Menzies said to me: "If there is a 'naming' centre, that is the seat of the lesion in this case."

As is well known to all who have given any attention to the complicated subject of aphasia, Broadbent has propounded the view, which is supported by Mills, that there exists, on the sensory side of the cerebral nerve apparatus, a special *naming* centre, and, on the motor side, a corresponding *propositioning* centre. Personally, I see no reason to adopt this view and to suppose that a "naming centre," other than the auditory speech centre, exists.

When this case first came under my notice, I was disposed to think that it was totally opposed to Broadbent's view. But more careful consideration has shown me that such is not necessarily the case; and although I see no reason whatever for believing in the existence of a "naming" centre (as distinct from the ordinary auditory speech centre), I do not now maintain, as I was at one time disposed to do, that the facts of this case entirely disprove the existence of a "naming" centre. For if a special "naming" centre exists, as Broadbent and Mills suppose, on the sensory side of the cerebral nerve mechanism, it is obvious that the nervous impulses which are generated in that centre must necessarily pass out through the motor-vocal speech centre in order that they may be vocalised; and consequently that an inability to name objects and persons may be due either to (*a*) destruction of the "naming" centre; (*b*) destruction of the nerve fibres which pass from that "naming" centre to the motor-vocal speech centre; or (*c*) destruction of the motor-vocal speech centre by which these impulses are emitted.

Now, in this case, in which the motor-vocal speech centre (Broca's convolution) was acutely and completely destroyed, there was, after the immediate effects of the lesion passed off (five days after the onset of the lesion), no motor-vocal aphasia, except the inability to name objects and persons (if that was, in this case, a motor defect).

But there is one difficulty in the way of accepting the view that the inability to name objects and persons was a motor defect, viz., that the motor-vocal speech centre was completely destroyed and yet there was no motor aphasia, except for the emission of names (if the inability to name objects and persons was a motor defect). In other words, if inability to name persons and objects was a motor defect and was due to the destruction of Broca's convolution, the destruction of that centre, since it was complete, should have produced inability to use *all* words (words other than names), *i.e.*, should have produced complete motor aphasia. Or, to put it another way, if the absence of motor aphasia (other than the inability to name objects and persons) was due (as I maintain) to the right motor-vocal speech centre taking up and carrying on the function of the motor-vocal speech centre which was destroyed (Broca's convolution), why was the right motor-vocal speech centre not also able to emit the names of objects and persons?

It may of course be said that the naming of objects and persons is a more elaborate (more highly specialised and less automatic) mental speech-process than the production of ordinary words (other than names); and that it is consequently conceivable that the right motor-vocal speech centre may be sufficiently educated and active to emit ordinary words (other than names), but not sufficiently educated and active to emit names. But such a supposition seems to me to be extremely difficult to accept, for I see no reason whatever why a motor speech-centre which can emit the impulses (*i.e.*, send to the ordinary psycho-motor centre for the lips, tongue, larynx, etc., the nervous impulses) concerned in the production of ordinary words should not be able to emit the impulses concerned in the production of the names of objects and persons. Further, as everyone knows, in many cases of motor-aphasia in which the motor aphasia is complete, the patient still retains the power of spontaneously uttering his own name and his wife's name—the names which by long practice have become, as it were, automatic. In cases of complete motor aphasia due to destruction of Broca's convolution, the few words which the patient retains (such as his own name and certain emotional and automatic utterances) are probably, I think, emitted by the right motor-vocal speech centre. But in this particular case the patient, when I saw him, could not name himself, and during the subsequent progress after I saw him was unable to spontaneously name his wife or his doctor. Though there was practically no other motor aphasic defect, he was unable to utter the very words (such as his own name) which are usually retained by patients affected with motor aphasia. It is consequently difficult, I think, to accept the view that in the case which I am at present recording the inability to name objects and persons was a motor defect, more especially since, as I have remarked in recording the case, the patient could *repeat* his own name, his wife's name and his doctor's name when they were mentioned to him, although he could not spontaneously emit them. The fact that the patient could repeat names which he could not spontaneously utter seems to show that the inability to name objects and persons was due to a defect on the sensory and not on the motor side of the speech mechanism.

Whether it is possible that a name or a word, which cannot be *spontaneously* emitted in consequence of complete destruction of Broca's convolution, can be *repeated* and emitted by the ordinary psycho-motor centre for the lower face, lips, tongue, larynx (*per* the lower end of the left ascending frontal convolution, *i.e.*, without having previously passed through the motor-vocal speech centre—Broca's convolution), or not, I do not venture to give an opinion. But this occurs to me as a possibility; the point deserves consideration and should be kept in view (with the object of having this possibility proved or disproved) in dealing with cases of motor aphasia.[1]

The most probable explanation of the inability to name objects and persons is, it seems to me, to suppose that the action of that part of the auditory speech-centre in which names (highly specialised parts of speech) are stored and recalled (or, as Broadbent and Mills would say, of a special centre other than the auditory speech-centre—the "naming" centre, as they term it) was deranged by the lesion in the motor-vocal speech centre, just as I have presumed that the action of the visual speech centre was temporarily deranged by that lesion. In other words, that the inability to name objects and persons was produced in the same *indirect* way in which I suppose the slight word-blindness was produced, and in which the more marked agraphia was, perhaps, produced. But here again there is a difficulty, for when I saw the patient he could write his own name, although he could not say it.

I see no reason to suppose that the inability to name objects and persons was due to a lesion of the conducting tract, which is supposed by some authorities to pass from a special centre, the "naming" centre, to the motor-vocal speech centre.

This seems to me sufficiently proved by the facts :—(*a*) the lesion was practically speaking confined to Broca's convolution and to the anterior end of the left island of Reil; (*b*) these parts were completely destroyed by the lesion; (*c*) the ordinary "way out" for names, and words other than names, was blocked in the emissive centre (Broca's convolution); it is therefore quite unnecessary and superfluous to suppose that a lesion existed in the tracts proceeding to that centre; (*d*) there was no evidence of a lesion in any tract proceeding from the supposed "naming" centre (or from the auditory speech-centre, in which I believe the so-called naming centre is situated and of which I believe it to be a component part) to the motor-vocal speech centre (unless that tract passes immediately beneath the anterior end of the island of Reil); and (*e*) that the emissive tract for names was obviously open (unblocked), for the patient could repeat a name (such as his own name or his doctor's name) which he could not spontaneously utter.

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[1] Since writing the above, I see that Joseph Collins makes a similar suggestion with regard to "echo" speech. He says :—"Whether such reflex or echo-like words cannot be produced by the action of sensory impulses directly on the central executive motor speech mechanism, I have often debated."—"The Faculty of Speech," by Professor Joseph Collins, page 170.

Since the left motor-vocal speech centre (Broca's convolution) was in this case completely destroyed, it seems to me that names as well as other words must in all probability (when pronounced in this way, *i.e.*, repeated) have been emitted through the opposite *right* motor-vocal speech centre.

From these statements it will be apparent that I have very great difficulty in giving a satisfactory explanation of the patient's inability to name persons and objects.

Conclusion.

It may of course be argued that the motor-vocal speech centre is not necessarily limited to the posterior end of the lower (third) left frontal convolution (Broca's convolution), or indeed to the adjacent parts of the lower (third) left frontal convolution (for in this case the softening involved not only the "foot," but also the "cap" and the "orbital portion" of the third left frontal convolution), but that it may, in exceptional cases, of which this was an example, be situated in the adjacent parts of the left hemisphere (the lower end of the left ascending frontal convolution, &c.). In other words, that the modern view, that the lower end of the left ascending frontal convolution is merely the ordinary psycho-motor centre for the lower face, tongue, larynx, &c., and that it is not, properly speaking, a part of the motor-vocal speech centre, is erroneous.

If this be granted, it must of course be allowed that it is unnecessary to suppose that the action of the left motor-vocal speech centre was in this case taken up and carried on by the corresponding centre in the right hemisphere.

But such a supposition seems to me, in the face of our modern knowledge (and especially in the light of Elder's case to which I have previously referred), altogether less likely than the theory which I have advanced. Further, Barlow's case *conclusively proves* that the right motor speech-centre *can* (after a short time) take up and perfectly carry on the motor-vocal speech function; and other arguments and facts can be adduced to show that the speech centres and speech faculties are, in some degree at all events, bilaterally represented in the cerebral hemispheres (see my lectures in the *Edinburgh Medical Journal*, and Bastian's "Lumleian Lectures on Aphasia").

In short, after the most careful and impartial consideration of the whole question, the most reasonable explanation of the absence of motor-vocal aphasia in this case, in which Broca's convolution was completely and acutely destroyed, seems to me to be that the function of the left motor-vocal speech centre was taken up and carried on by the motor-vocal speech centre in the right hemisphere of the brain.

DESCRIPTION OF FIGURES.

FIG. 1.

The left hemisphere of the brain in the case of aphasia described in the text, showing the exact extent of the lesion.

The degenerated areas are shaded in black.

The figures point to the parts (convolutions) which are correspondingly numbered in the horizontal sections (see figs. 3, 4, 5 and 6).

The letters RO. are placed immediately above the upper end of the fissure of Rolando.

The letter S. is placed at the commencement of the fissure of Sylvius, at the point at which the horizontal and ascending limbs (marked by white lines) are given off.

FIG. 2.

The left hemisphere of the brain in the case of aphasia described in the text, showing the exact position of the series of longitudinal (horizontal) sections which were made through it.

The figures 1 to 6 point to the different sections, the upper surfaces of which were photographed, and are reproduced in figs. 3, 4, 5 and 6. The under surface of section 5 is reproduced in fig. 7.

FIG. 3.

Longitudinal (horizontal) section of the brain in the case of aphasia described in the text (the upper surface of section 2 in fig. 2.)

A small area of softening, which corresponds to the uppermost point of Broca's convolution, is shaded black.

1 = Posterior end of the second left frontal convolution.
2 = The uppermost point of Broca's convolution, softened and degenerated.
3 = The left ascending frontal convolution.
4 = The left ascending parietal convolution.
2 R. F. = The second right frontal convolution.
R. A. F. = The right ascending frontal convolution.
R. A. P. = The right ascending parietal convolution.

FIG. 4.

Longitudinal (horizontal) section of the brain in the case of aphasia described in the text (the upper surface of section 3 in fig. 2). The area of softening, which is shaded in black, involves Broca's convolution and the anterior end of the left island of Reil. These parts were completely destroyed.

5 = First left frontal convolution.
6 = Second left frontal convolution.
7 = Anterior end of the "cap" of Broca's convolution.
8 = The "cap" of Broca's convolution.
9 = The "foot" and "ascending portion" of Broca's convolution.
10 = Left ascending frontal convolution.
11, 11 = Left ascending parietal convolution.
12 = The anterior end of the left supra-marginal convolution.

The letter R. is placed on the white matter just beneath the anterior end of the left island of Reil.

R. 3 F., R. 3 F. = Right third frontal convolution.
R. A. F. = Right ascending frontal convolution.
R. A. P. = Right ascending parietal convolution.

FIG. 5.

Longitudinal (horizontal) section through the brain in the case of aphasia described in the text (the upper surface of section 4 in fig. 2).

A large area of softening and destruction, which is shaded in black, is seen in the position of the posterior and middle parts of the third left frontal convolution, and of the anterior end of the left island of Reil.

13 & 14 = Second left frontal convolution.
15 = Third left frontal convolution; a small gyrus which was practically unaffected.
16 = "Cap" of Broca's convolution.
17 = "Foot" and "ascending portion" of Broca's convolution.
18 = Left ascending frontal convolution.
19 = Left ascending parietal convolution.

R. 3 F., *R. 3 F.* = Right third frontal convolution.
R. A. F. = Right ascending frontal convolution.
R. 1 T. S. = Right first temporo-sphenoidal convolution.

Note.—The section of the right hemisphere is at a slightly lower level than that of the left.

FIG. 6.

Longitudinal (horizontal) section through the brain in the case of aphasia described in the text (the upper surface of section 5 in fig. 2).

The area of softening and destruction, which is shaded in black, involves the anterior (orbital) portion of the lower (third) left frontal convolution.

20 & 21 = Second left frontal convolution.
22 = Orbital part of the third left frontal convolution.
23 = Anterior end of the first left temporo-sphenoidal convolution.

FIG. 7.

The inferior surface of the frontal lobes of the brain in the case of aphasia described in the text (the under-surface of section 5 in fig. 2).

The portion of the lower (third) left frontal convolution which is shaded black, and to which the figure 24 points, was softened and degenerated.

NOTE ON THE UPPER TERMINATIONS OF THE DIRECT CEREBELLAR AND ASCENDING ANTERO-LATERAL TRACTS.

BY ALEXANDER BRUCE, M.A., M.D., F.R.C.P.E., F.R.S.E.

CASES of early degeneration of these tracts in man do not appear to present themselves very frequently for examination at a stage when Marchi's method can be employed with success in tracing these to their ultimate termination; and since, in spite of the experimental work in animals done by Loewenthal, Mott, Tooth, and others, there still exists considerable difference of opinion on this subject, the following personal observations may not be superfluous.

In a case in which a sarcomatous tumour had compressed the spinal cord in the lower cervical region, recent ascending degenerations were found in the posterior columns on both sides, and in the lateral column of the left side, while an older degeneration, which did not yield any, or merely a minimal number of black granules when treated by Marchi's method, existed in the ascending tracts in the lateral columns of the right side.

The cord, medulla, cerebellum, and cerebral peduncles were impregnated in the usual manner with Marchi's solution after previous hardening in Müller's fluid.

I.—The postero-median columns of both sides showed a complete degeneration as high as the nucleus gracilis. This degeneration ended entirely in the nucleus, and did not extend either into the fillet, the restiform body (as stated by Thomas, *Le Cervelet*), or the tract of Gowers (as recently stated by Rossolymo, *Neur. Centrabl.*, October 15, 1898).—(Figs. 1 and 2).

II.—There was a partial degeneration in the postero-external column. This affected its inner portion from the periphery to the posterior commissure, leaving the outer portion quite intact. The healthy outer portion was evidently formed by ascending fibres from roots which had entered above the lesion and which, in accordance with

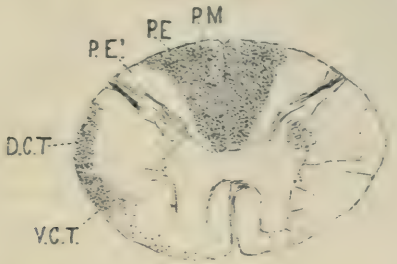


FIG. 1.

Transverse section of the cord at the level of the second cervical segment.

P. M. = Postero-median column.
P. E. = Degenerated portion of postero-external column.
P. E'. = Undegenerated ,, ,, ,, ,,
D. C. T. = Direct cerebellar tract (or dorsal cerebellar tract).
V. C. T. = Ventral cerebellar tract.

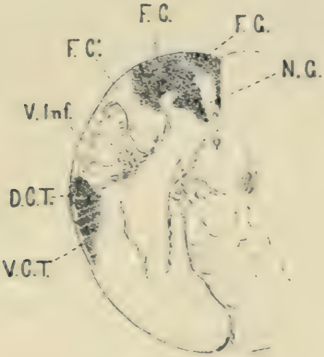
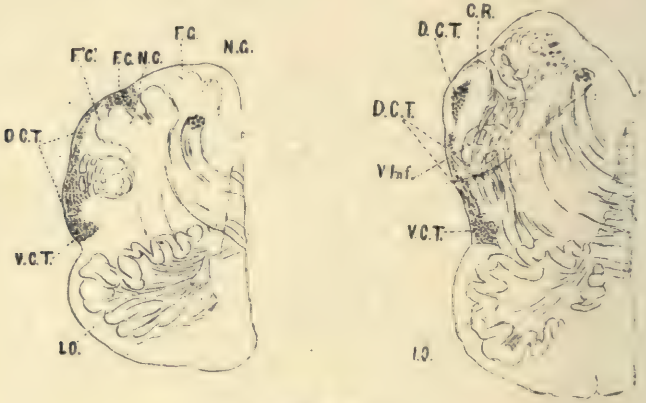


FIG. 2.

Transverse section through lower medulla.

F. G. = Funiculus gracilis.
N. G. = Nucleus gracilis.
F. C. = Degenerated portion of funiculus cuneatus.
F. C'. = Undegenerated ,, ,, ,, ,,
D. C. T. = Dorsal cerebellar tract.
V. C. T. = Ventral cerebellar tract.

Singer's law, had pushed the degenerated ascending fibres inward (fig. 1). The distribution of the degenerated and normal fibres within the funiculus cuneatus in the medulla remained the same as in the postero-external column of the cord, the degenerated ones occupied its inner half, and the healthy ones the outer. The fibres which entered the nucleus also maintained the same relationship (fig. 2), no degenerated fibres being found in its outer half. As in the case of the postero-median column, not a single degenerated fibre was traceable directly into the restiform body or fillet, or to any other point beyond the nucleus. We may, I think, fairly conclude that the outer half of the funiculus cuneatus also terminates completely in the nucleus.



FIGS. 3 & 4.

Transverse section through middle medulla.

N. C. Nucleus cuneatus.
I. O. = Inferior olive.
V. Inf. = Inferior root of the fifth nerve.

Other letters as in Fig. 2.

III.—The direct or dorsal cerebellar tract could be traced upwards along the lateral margin of the lower third of the medulla in the position usually assigned to it. In front of it lay the ventral cerebellar tract (fig. 2). The dorsal cerebellar tract passes backwards superficially to the inferior root of the fifth nerve (fig. 3) to its position in the restiform body, within which it occupies the central core (fig. 4), as was figured in Mid and Hind Brain (plate IX). After entering the cerebellum it ascends in front of the nucleus dentatus, and, at the upper extremity of the latter passes backwards along the convex margin of the superior cerebellar peduncle immediately after that structure has emerged from the hilum of the nucleus dentatus (fig. 6). At the posterior margin of the peduncle the cerebellar tract bends inwards towards the superior vermis, and terminates *on both sides of the lobulus centralis, the lobulus monticuli, and*, to a slight extent also, *in the lobulus lingualis.* The majority of the fibres terminate in the same side of these lobules, in the manner indicated in fig. 6; but a very *considerable number cross the mesial plane in the great anterior or superior decussating commissure of the cerebellum of Stilling*, and end in the cortex of the opposite side of the above-mentioned lobules.

The superior cerebellar commissure would appear to be mainly composed of decussating fibres from the two cerebellar tracts, and is therefore not to be regarded as analogous to the corpus collosum of the cerebral hemispheres, as some authors have stated. Thus the main termination of the dorsal cerebellar tract is partly direct and partly crossed in the upper portion of the superior vermis. A small number of degenerated fibres, after rounding the posterior margin of the peduncle, bend sharply downwards internal to the posterior part of the nucleus dentatus, and seem to reach the nodulus and uvula of the inferior vermis. The limited number and sparse distribution of these degenerated fibres do not permit one to determine whether or not any of them crossed to the opposite side. No distinct evidence could be found that any fibres terminate in any of the nuclei of the middle lobe, and it was quite certain that none enter the nucleus dentatus or the cortex of the cerebellar hemispheres.

IV.—The ventral cerebellar tract does not become completely disengaged from the dorsal until nearly the middle of the medulla. Below this it lies in front of the dorsal tract, forming a circumscribed bundle close behind the olive. Above, it lies first to the inner side of and then just behind the lateral angle (fig. 5) of the medulla. In the pons it penetrates the corpus trapezoides on the outer side of the superior olive, ventral to the nucleus of the facial nerve (figs. 6 and 7), and internal to its emerging root. Here (fig. 6), its fibres become cut somewhat obliquely, as they begin to pass outwards and backwards. They pass in front of the sensory root of the fifth nerve (fig. 8), and reach the periphery of the pons as it emerges from the

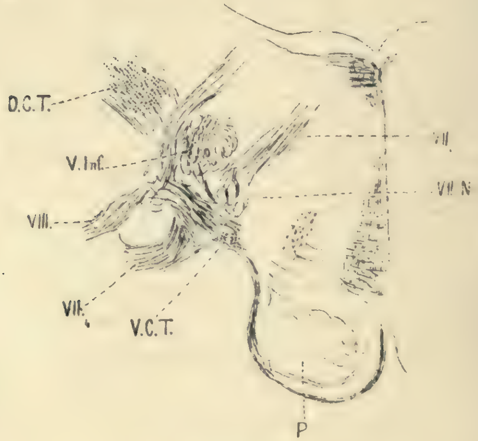


FIG. 5.

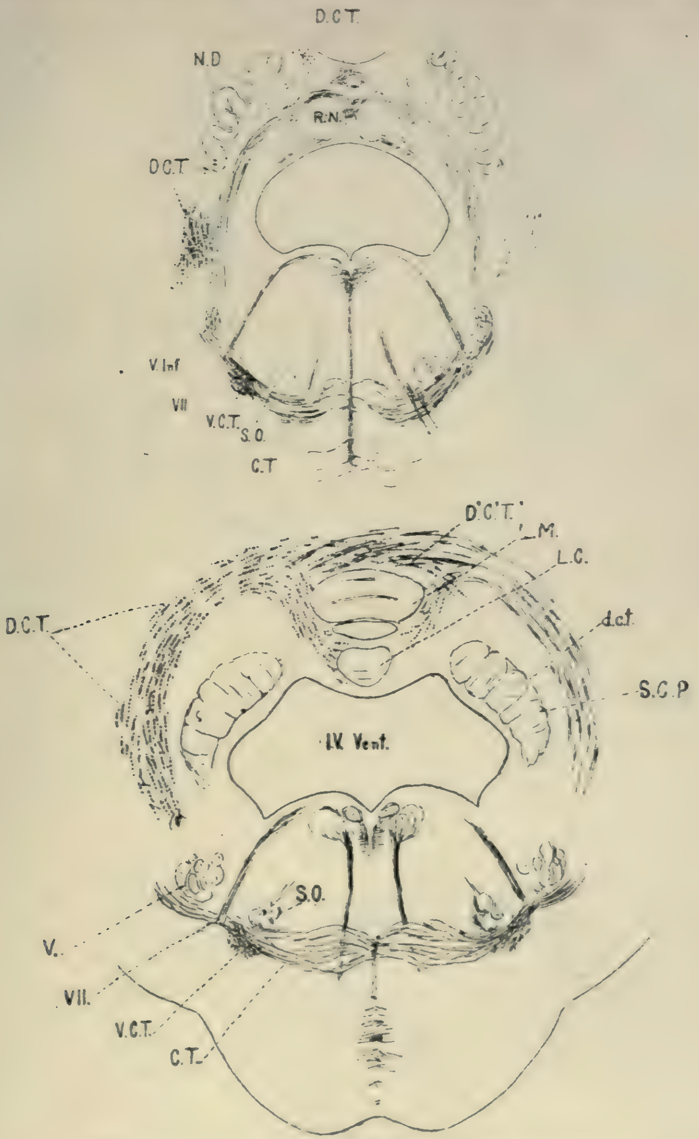
Transverse section through upper medulla.

P. = Pyramid.
VII. N. = Nucleus of seventh nerve.
VII., *VII.*$_1$, *VII.*$_4$ = Parts of seventh nerve.
VIII. = Eighth nerve, vestibular root.

Other letters as in preceding figures.

Note.—The black dots in D.C.T. and V.C.T. are indicated too faintly.

cerebellum. Here they lie just external to the anterior part of the lateral fillet. [In fig. 8 a few of the uppermost fibres of the direct cerebellar tract come into view, and suggest an appearance of continuity with the ventral cerebellar tract.] From this point they curve along the outer side of the superior cerebellar peduncle in an upward and backward direction, then inward round its posterior margin, and then



FIGS. 6 & 7.

Transverse section through lower pons and cerebellum.

N. D. = Nucleus dentatus.
S. O. = Superior olive.
C. T. = Corpus trapezoideum.
S. C. P. = Superior cerebellar peduncle.
D'.C'.T'. = Crossed degenerated dorsal cerebellar tract.
d. c. t. = Healthy dorsal cerebellar tract from opposite side.
L. M. = Lobulus monticuli.
L. C. = Lobulus centralis.
V. = Fifth nerve nucleus.

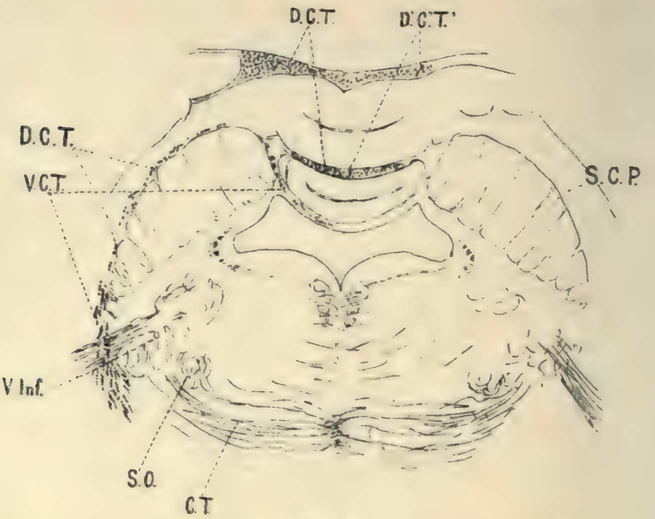


FIG. 8.

Section through pons at the level of the emerging fifth nerve.

Letters as before.

V. C. T. The ventral cerebellar tract is seen rounding the root of the V. nerve, and becoming apparently continued in a backward direction along the outer aspect of the superior cerebellar peduncle, into the anterior medullary velum. The intermediate portion, however, belongs (*D. C. T.*) to the dorsal cerebellar tract.

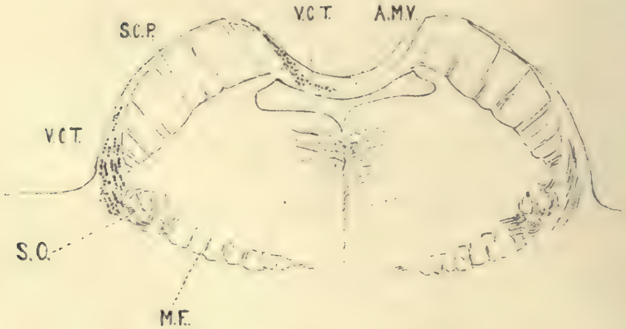


FIG. 9.

Section through pons immediately above the nucleus of the fifth nerve.

V. C. T. The ventral cerebellar tract in two positions near the lateral fillet, external to the superior olive, and again in the anterior medullary velum (*A. M. V.*).

M. F. = Median fillet.

inwards and downwards towards the anterior medullary velum (figs. 8, 9, 10). The description just given corresponds absolutely with that given by Mott as the results of experimental section of the tract in the cord of the monkey. The degenerated tract was rendered quite visible to the naked eye after the part had been fourteen days in Müller's fluid, and I regret that I did not have it drawn before cutting up the tissue into thin slices for immersion in Marchi's solution. It ended in the lingual lobule of the vermis, and almost entirely on that of the same side. There was some indication that a few of its fibres entered the inferior corpus quadrigeminum on the same and on the opposite side, but the number of fibres which did so was altogether insignificant compared with the number which entered the lingual lobule.

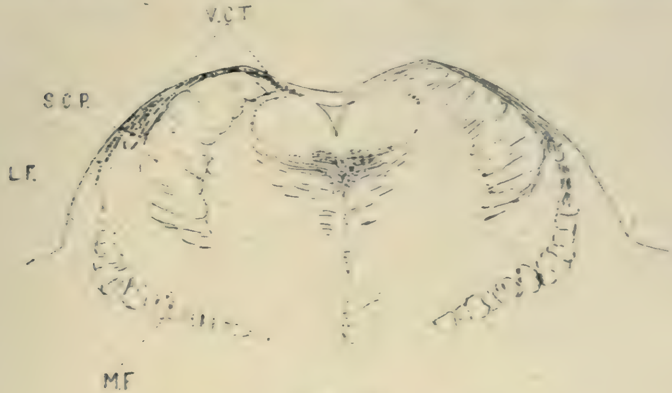


FIG. 10.

Section at a level slightly higher than that of Figure 9.

V. C. T. The ventral cerebellar tract is now seen curving round the posterior extremity of the superior cerebellar peduncle.

From the above description it is evident that the ventral cerebellar tract has in man the same course and termination which have been assigned to it in animals by the observations of Loewenthal, and, in this country, of Mott and Tooth. I am not in a position to state definitely whether any of the fibres of the tract ended in the lateral nucleus of the medulla. Some of them passed through that nucleus, but I could not say whether the number of degenerated fibres was less on its upper than on its under side. Unless there is a very distinct diminution in the number of degenerated fibres at two different levels, one cannot be certain that that diminution is not due to some accident in the penetration of the Marchi's liquid. Therefore I hesitate to state definitely that any of the tract terminates in this nucleus, although it is extremely probable.

ON THE DORSAL OR SO-CALLED SENSORY NUCLEUS OF THE GLOSSOPHARYNGEAL NERVE, AND ON THE NUCLEI OF ORIGIN OF THE TRIGEMINAL NERVE.

BY ALEXANDER BRUCE, M.A., M.D., F.R.C.P.E., F.R.S.E.
Lecturer on Pathology, School of Medicine, Edinburgh, &c.

THE origin of the glossopharyngeal and vagus nerves are for the most part stated as being, firstly, a motor nucleus, the nucleus ambiguus; secondly, a sensory nucleus, or dorsal nucleus lying to the posterior or outer side of the hypoglossal nucleus; and thirdly, the nucleus of the descending root or fasciculus solitarius. With regard to the nucleus ambiguus there is practically no doubt that this is motor in function. There may be considerable difference of opinion as to whether the nucleus sends fibres to the nerves on both sides or only to that of its own side; but it is universally admitted to be a motor nucleus. In like manner, with regard to the descending root or fasciculus solitarius, there is now a practical unanimity among neurologists that this is sensory in function, and that its fibres terminate in relation to cells in the gelatinous substance situated in immediate proximity to it.

The dorsal or so-called sensory nucleus has up to a recent date been generally assumed to be, as the name implies, sensory in nature, although this view has not been universally accepted. While Kölliker, Cajal and Van Gehuchten are agreed that it is sensory, on the other hand, observers like Eisenlohr, Dees, Forel, Homen and Marinesco maintain that the nucleus is motor and not sensory. In Kölliker and Naegeli's *Festschrift*, 1892, Forel discovered that, when the glossopharyngeal nerve of the newly-born guinea-pig was divided, the cells of the so-called sensory nucleus atrophy and disappear completely. On the strength of Gudden's law he argues that the cells would not have disappeared had they not been the cells of origin of the fibres, and, therefore, that they must be motor or efferent and not sensory in their nature. In contrast to this, while the fibres of fasciculus solitarius had also degenerated, the cells in the gelatinous substance in relation to which these fibres terminate were practically unaltered, being merely approximated to each other by the disappearance of the terminal fibres which had lain between them. This paper passed almost unnoticed until, in 1897, Marinesco published in the *Comptes rendus de la Société de Biologie* a short but important article on the result of the examination of the nuclei by Nissl's method, shortly after section of the glossopharyngeal nerve. He ascertained that six days after section "reaction à distance" had begun in the dorsal nucleus. In fifteen days the reaction was complete. It is now well known that this phenomenon only occurs in cells of the same neuron as the divided fibres, and that it does not appear in the nuclei which are not directly connected with the nerve fibres. Therefore he argues that if the root passing to this nucleus were sensory in function, it would not have been directly connected with the cells of the nucleus, and that they would not have undergone the degeneration mentioned. Marinesco also found that the nucleus ambiguus showed the same reaction, and has put forward the ingenious theory that the nucleus ambiguus is the motor centre for the striped muscles supplied by the vagus and glossopharyngeal nerves, while the small-celled dorsal nucleus is the centre for the non-striped muscles.

The opportunity has recently been afforded me of examining a case in which a tumour, after destroying the fifth nerve, had completely involved the glossopharyngeal nerve within the jugular foramen, but had spared the vagus nerve. The medulla was carefully prepared by Marchi's method and cut into fine sections, with the result that complete corroboration was given of the views of Forel and Marinesco. It was found that while the whole of the glossopharyngeal

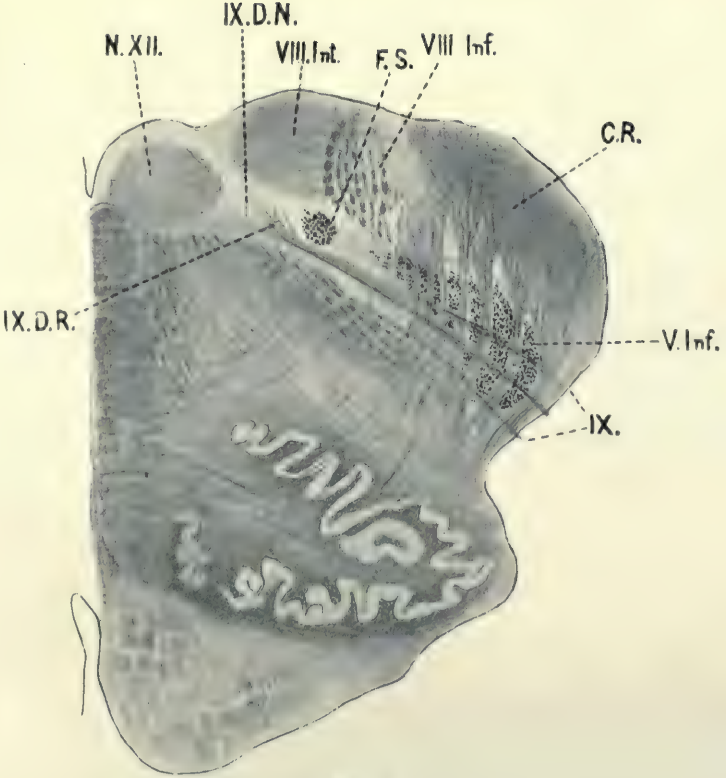

IX.D.N.
N. XII.
VIII.Int.
F.S.
VIII Inf.
C.R.
IX.D.R.
V.Inf.
IX.

FIG. 1.

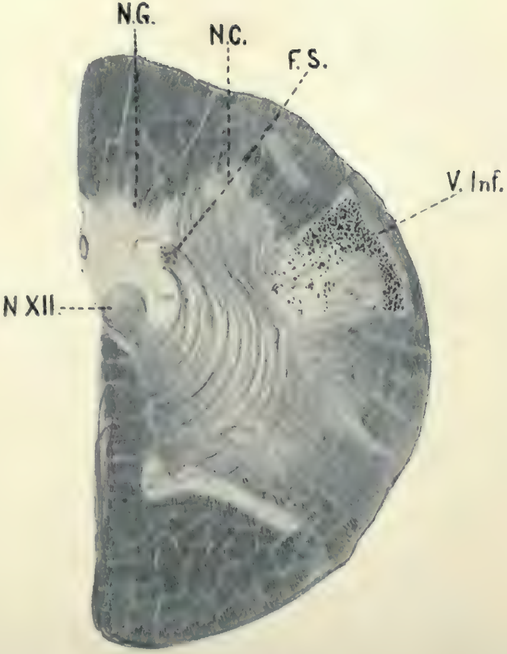

N.G.
N.C.
F.S.
V. Inf.
N XII.

FIG. 2.

fibres which enter the upper part of the fasciculus solitarius were degenerated, not a single degenerated fibre could be traced towards the dorsal nucleus or to the nucleus ambiguus. The appearance presented is indicated in fig. 1. This condition of the fibres entering the dorsal nucleus obtained in all the sections involving the glossopharyngeal nucleus, so that there could be no manner of doubt that the nerve roots connected with it were not afferent, but efferent in character.

The fibres of the fasciculus solitarius which were found to be degenerated at its upper extremity gradually disappeared in a downward direction, obviously because they terminated in relation to the cells of the gelatinous substance, while healthy fibres, belonging no doubt to the vagus nerve, entered below, so that in its lower two-thirds the main portion of the fasciculus was formed by undegenerated fibres (fig. 2).

We may conclude, therefore, that the fasciculus solitarius is an afferent nerve belonging, in its upper part, to the glossopharyngeal nerve, and in its middle and lower part mainly to the vagus nerve: and that the dorsal nucleus and the roots from it are motor, as suggested by Forel and Marinesco, and not sensory.

The examination of the degenerations which followed the destruction of the fifth nerve showed that:—

(1) The descending or (as I prefer to term it) the superior root was intact. Therefore we may assume that this root is efferent and not sensory. This is in harmony with the recent experiment of Ferrier and Turner.

(2) The motor root was intact.

(3) No degenerated fibres were traceable into the locus cœruleus of the same or of the opposite side.

(4) The sensory root was entirely uncrossed. Its fibres terminated in the sensory nucleus of the same side, and also passed downwards in the ascending (or inferior) root. The vast majority of these lay on the external aspect of the gelatinous substance of Rolando, but a considerable proportion passed downwards on the inner aspect of this structure, corresponding to what I described in my "Illustrations of Mid and Hind Brain," in 1892, as the inner descending root.

[Permission was not granted for the examination of the spinal cord, so that I was unable to determine the lower limit of the degeneration in this root.]

The analogy between the nuclei of origin of the fifth nerve and those of the vago-glossopharyngeal nerve is very striking: each has a motor nucleus; each has a descending sensory root; and each has a nucleus composed of cells different in character from those of the motor nucleus but giving origin to efferent fibres. It is true that the cells of the upper root of the fifth nerve are not of the same character as those of the dorsal nucleus of the vago-glossopharyngeal nerve. But in other respects the similarity is very close.

This similarity leads one to enquire whether this threefold origin may not be the primitive arrangement for the cranial nerves—a sensory root, a motor root to striped muscle, and an efferent root (to non-striped muscle ?). This arrangement has persisted in the two sets of nerves under consideration, but has disappeared more or less completely in the remaining cranial nerves. In the third nucleus, however, we have the remarkable postero-internal group of cells (Edinger-Westphal nucleus) which differs from the remainder of the nucleus in the character of its cells and in its pallor in Weigert Pal sections—points of resemblance to the dorsal vago-glossopharyngeal nucleus. There is some reason to think that this nucleus supplies the non-striped muscles of the iris. Again, within the hypoglossal nucleus there is at one point a small circumscribed group of cells, somewhat similar in character to the postero-internal group in the third nucleus, which may be a remnant of this efferent nucleus.

The facial nerve has been so modified that only a motor element is now detectable, and the same may be said of the sixth and fourth nerves. In the lower vertebrata, however, it may be possible to find traces of the missing element in these.

Whether further search will confirm the hypothesis of this mode of origin of the cranial nerves, it may now, I think, be regarded as certain, on the ground of the experiments of Forel, Marinesco, Ferrier and Turner in animals, and my case of pathological destruction of the nerves in man, that both the dorsal glossopharyngeal nucleus and the nucleus of the upper root of the trigeminal nerve give origin to fibres which are efferent.

DESCRIPTION OF FIGURES.

FIG. 1.

Transverse section of upper medulla, prepared by Marchi's method.

N. XII. = Nucleus of hypoglossal nerve.
IX. D.N. = Dorsal nucleus of glossopharyngeal nerve.
VIII. Int. = Internal nucleus of auditory nerve.
F.S. = Fasciculus solitarius, or descending root of glossopharyngeal nerve, in a state of degeneration.
IX. D.R. = Root of glossopharyngeal nerve from dorsal nucleus, undegenerated.
C.R. = Corpus restiforme.
V. Inf. = Inferior root of nerve.
IX. = The glossopharyngeal nerve.

FIG. 2.

Transverse section of the lower medulla.

N.G. = Nucleus gracilis.
N.C. = Nucleus cuneatus.
F.S. = Fasciculus solitarius, degenerated only slightly, contains healthy fibres from vagus.
V. Inf. = Inferior root of V nerve, showing degenerated fibres on the inner side as well as on the outer side of the gelatinous substance of Rolando.

EXPERIMENTAL OBSERVATIONS ON EARLY DEGENERATIVE CHANGES IN THE SENSORY END ORGANS OF MUSCLES.[1]

BY F. E. BATTEN, M.D.

Contents.

(1) Introduction.
(2) Object of the research.
(3) Plan of research.
  (*a*) Operative procedure.
  (*b*) Mode of preparation of tissue.
  (*c*) Microscopical examination.
(4) Summary of results obtained.
(5) Comparison of results with those of other observers.
(6) Conclusions.

(1) Introduction.

While examining the muscle-spindle under various pathological conditions,[1] I found that certain degenerative changes rendered apparent by the Marchi method, were present in the nerve termination in a case of tabes dorsalis, whereas the nerve passing to the end organ remained apparently normal, and as a possible explanation it was suggested that degenerative changes might take place at a point remote from the cell, the intermediate portion of the nerve remaining unaffected. It was, then, in the hope of being able to produce these appearances that the following series of experiments were undertaken, and although the results

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[1] The expenses of this research have been defrayed by a grant from the Scientific Grants Committee of the British Medical Association, and the microscopical examination performed in the Pathological Laboratory of the National Hospital, Queen Square.

hoped for have not been obtained, yet the experiments have yielded results sufficiently important to justify their being placed on record. Dogs were alone used and inasmuch as previous experience had taught me that the smaller the animal the more difficult it was to tease out the muscle-spindle, large animals were alone used.

I am indebted to Dr. Risien Russell for performing the operations, and our thanks are due to Professor Vaughan Harley for allowing the experiments to be conducted at the Laboratory for Chemical Pathology at University College, London.

Having in a recent paper dealt somewhat fully with the work of previous observers, I do not propose to repeat the historical digest there given, but I have under heading (5) discussed the results of those observers which are most nearly in relation to the present subject.

(2) OBJECT OF THE RESEARCH.

The two chief objects that I had in view when I undertook the experiments on the result of which this paper is based were:—(*a*) To ascertain whether degeneration of the part of the neuron most remote from the cell takes place before other parts of it suffer similarly. (*b*) To reproduce if possible in animals the changes which I have previously shown to be present in the terminal end organ in the case of tabes dorsalis in man.

(3) PLAN OF RESEARCH.

(*a*) *Operative Procedure.*—The animals were placed under the influence of ether administrated by inhalation, and anæsthesia thus induced was maintained throughout the operation. The hair was shaved from the neck, the skin thoroughly cleansed by means of soap and water, and then carefully disinfected by means of perchloride of mercury lotion (1-1,000). Dr. Russell then made a longitudinal incision in the neck, exposed the mixed nerves which enter into the formation of the brachial plexus, and by severing them close to their point of emergence from the neural-canal and where they combine to form the plexus, was enabled to remove large portions of the nerve trunks from the 5th cervical to the 1st dorsal inclusive. The edges of the skin wound were brought together by means of sterilised horsehair sutures, after all hæmorrhage had been arrested, and an antiseptic dressing then applied.

The animals were allowed to live for periods of twenty-four, forty-six, seventy-two, ninety-six, and one hundred and twenty hours and seven and fourteen days, at the end of which times each was killed by an overdose of chloroform administered by inhalation.

(*b*) *Mode of Preparation of Tissue.*—Immediately after the animal was killed the fore-limb was removed and the biceps muscle dissected out, this was cut into small pieces in the direction of the muscle fibre and placed into Sihler's solution No. 1. The skin was then removed from the remaining portion of the leg, and the whole placed into Muller's fluid.

From each biceps, after being treated by Sihler's method,[1] 12 or more spindles were teased out, of these 3 were mounted in the ordinary way in Canada balsam without further staining, 6 others were placed into Muller's fluid for fourteen days and then into Marchi's fluid for seven days, they were then washed in water for two days, and after being treated in the usual manner were embedded in celloidin and cut in a longitudinal direction in series. Of the spindles so treated two were mounted without further staining, while the other four were stained by Pal's method.

From the fore-limb, after hardening in Muller's fluid for four months, the supinator longus muscle was removed together with the musculo-spiral nerve portions from the muscle, and portions from the nerve, both near its entrance into the muscle and also high up in its course through the brachial plexus, were placed into Marchi's fluid for seven days, and after being treated in the usual manner they were embedded in celloidin and cut in a longitudinal and transverse direction.

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[1] For details of this method see original paper *Archiv. für. Anat. Physiog. Physiologische Abtheiling*, s. 202, 1895, or BRAIN, 1897, p. 151.

(*c*) *Microscopical Examination.*—I propose to deal with the various points in the following order :—

I. The muscle-spindle and the nerve termination as studied by Sihler's method (Table I.). (*a*) Under normal conditions; (*b*) At various times after section of nerve.

II. The muscle-spindle and the nerve termination as studied by (1) Marchi's method; (2) Pal's method (Table I., columns ii. and iii.); (*a*) Under normal conditions; (*b*) At various times after section of the nerve.

III. The condition of the nerve within the muscle, at its entrance into the muscle, and near its origin from the cord at various times after section of the nerve; (*a*) by Sihler's method; (*b*) by Marchi's method; (*c*) by Pal's method (Table II.).

I. The muscle-spindle and nerve termination in the dog as studied by Sihler's method.

(*a*) *Under Normal Conditions.*

The muscle spindle of the dog is as it is in other animals, spindle in shape, being generally about 3 to 4 mm. in length, though it is not easy to measure its actual length, as the sheath gradually becomes thinner and thinner, and the exact point of termination is difficult to fix. In breadth the muscle-spindle measures about ·16 mm. at its broadest part.

Two nerves as a rule enter the muscle spindle, the one —a nerve composed of fibres of varying size, but containing large fibres (·008 mm.)—enters the equatorial region, the other —a nerve containing fine fibres (·002-·004 mm.)—enters towards the pole. Fine, well striated muscle fibres, generally two, sometimes three in number, enter the spindle at one pole and pass through to the opposite pole; at the equatorial region these muscle fibres undergo division, varying greatly in size, the smallest measuring about ·004 mm. in diameter, others measuring ·016 mm. and ·024 mm. Towards the poles the fibres are more uniform in size, measuring about ·024 mm. (extra-fusal muscle fibres measure about ·05 mm.).

These fibres are enclosed in a sheath composed of fine laminæ with elongated nuclei, like the Henle sheath of a nerve, there being as a rule one or two layers at the pole and increasing to four or five layers at the equator.

The nerve bundle which enters the equatorial region gives off fine nerve fibres, which pass towards the poles of the spindle and lie parallel to the muscular fibres (plate III., fig. 1). The method of termination of these fibres in the dog I propose to deal with at some future time.

The same nerve bundle gives off large nerve fibres; these as a rule make a bend soon after entering the spindle and come to lie parallel to the intra-fusal muscle fibres at a point near the equatorial region of the spindle; the intra-fusal muscle fibre at this point is seen to have lost its transverse striation, and contains large, clear, hexagonal cells (plate I., figs. 2-5, etc.), which at one point completely fill the muscle fibre, but gradually tail off in both directions into extremely fine cells, which lie in the centre of the muscle fibre (plate I., fig. 5). These large hexagonal cells appear to have no nucleus, they stain feebly with hæmatoxyline (unless previously treated with Muller's fluid, in which event they stain well[1]); they completely interrupt the striation of the muscle fibre so long as they fill it, but as they tail off the striation again begins to appear, and becomes marked before the cells have completely tailed off (plate I., figs. 5 and 11). These cells measure from ·006 to ·008 mm. in diameter, and were first described by Kühne.[2] Around these cells the nerve fibre, or rather, the axis-cylinder, winds itself in a spiral form (plate I., figs. 2 and 5; plate II., fig. 2), the nerve seems sometimes to join the spiral in the centre of its course (plate I., fig. 2), sometimes towards the end; the spiral is not always perfectly regular in its coils (plate I., fig. 2), and usually passes for a short distance on to the striated portion of the muscle. The spiral occupies in the whole of its length about ·16 mm. (plate I., fig. 1); the nerve fibres composing the spiral are about ·002 mm. in diameter, and there are on the average 12 or 13 coils in the

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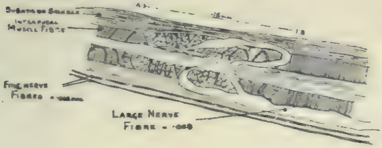
[1] See statement in BRAIN, 1897, p. 157.

[2] It is not improbable that the method of preparation is in part responsible for the shape of these cells, which are swollen up by the action of the acetic acid; I am of opinion that they are in reality nuclei which are aggregated at this point in the muscle fibre.

PLATE I.

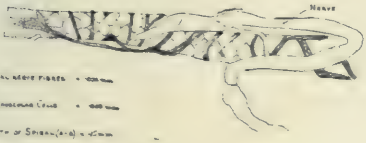
NORMAL SPINDLE FROM A DOG TO SHOW SPIRAL NERVE TERMINATION AND JUNCTION OF NERVE FIBRE WITH SPIRAL

FIG. 1



THE SAME UNDER $\frac{1}{12}$ IMMERSION

FIG. 2



Spiral nerve fibres = [illegible] mm

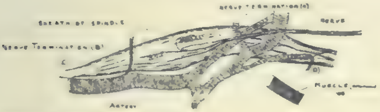
Intramuscular Cells = [illegible] mm

Length of Spiral (A–B) = [illegible] mm

Large Nerve Fibre = ·008

NORMAL MUSCLE SPINDLE FROM A DOG TO SHOW POSITION OF SPIRAL NERVE TERMINATION

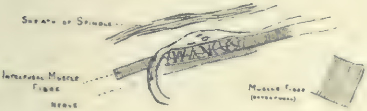
FIG. 3



Length of Spindle (C to D) = 1·275 mm

NERVE TERMINATION (A)

FIG. 4



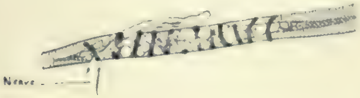
Length of Spiral = ·12 mm

Breadth of Spiral = ·010 mm

Intramuscular cells = [illegible] – [illegible] mm

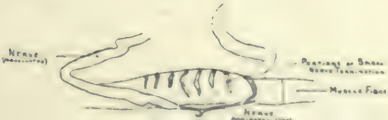
NERVE TERMINATION (A) TO SHOW INTRAMUSCULAR NUCLEI ENCIRCLED BY A SPIRAL NERVE FIBRE. THE STRIATION OF THE MUSCLE FIBRE IS INTERRUPTED.

FIG. 5



NERVE TERMINATION 24 HOURS AFTER SECTION OF THE NERVE
DOG IN LEFT BICEPS

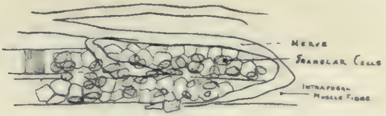
FIG. 6



NERVE TERMINATION 48 HOURS AFTER SECTION OF THE NERVE TO SHOW DISAPPEARANCE OF SPIRAL AND INCREASE OF GRANULAR CELLS. DOG [illegible] LEFT BICEPS

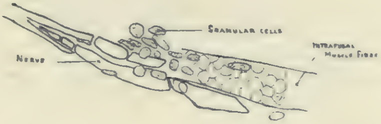
Large cells = [illegible] Granular cells = [illegible] ·006 Nerve fibre [illegible]

FIG. 7



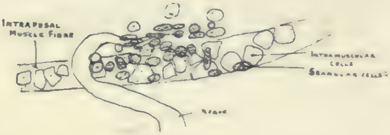
NERVE TERMINATION 72 HOURS AFTER SECTION OF THE NERVE

FIG. 8



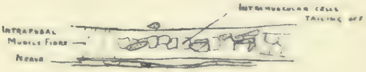
NERVE TERMINATION 4 DAYS AFTER SECTION OF THE NERVE
DOG IV LEFT BICEPS

FIG. 9



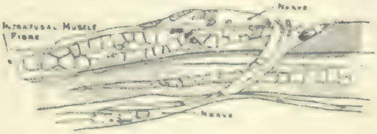
NERVE TERMINATION 5 DAYS AFTER SECTION OF THE NERVE DOG IV RIGHT BICEPS

FIG. 10



NERVE TERMINATION 7 DAYS AFTER SECTION OF THE NERVE
INTRAMUSCULAR CELLS = ·006 mm DOG [illegible] RIGHT BICEPS

FIG. 11



NERVE TERMINATION [illegible] DAYS AFTER SECTION OF NERVE DOG [illegible] R BICEPS

FIG. 12

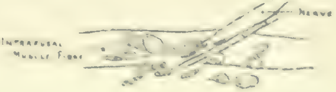


PLATE II.

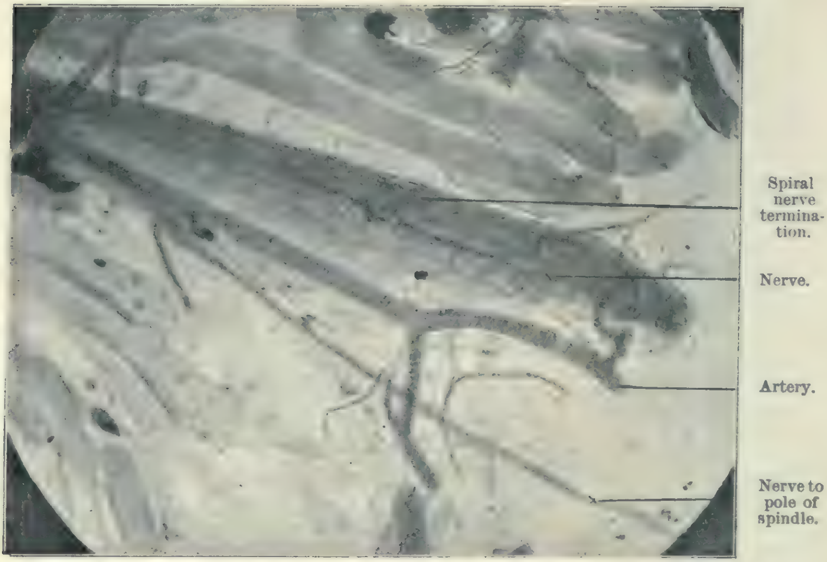


FIG. 1.—Photograph of a normal muscle spindle from a dog, showing two spiral nerve terminations. The lower one is clear, the upper one lies under the nerve and not so distinct. The nerve can be seen entering from the left, and before reaching the spiral divides into two. Above the spindle is seen an artery, and above the artery another nerve passing to the pole of the spindle. Magnified 70 diameters.

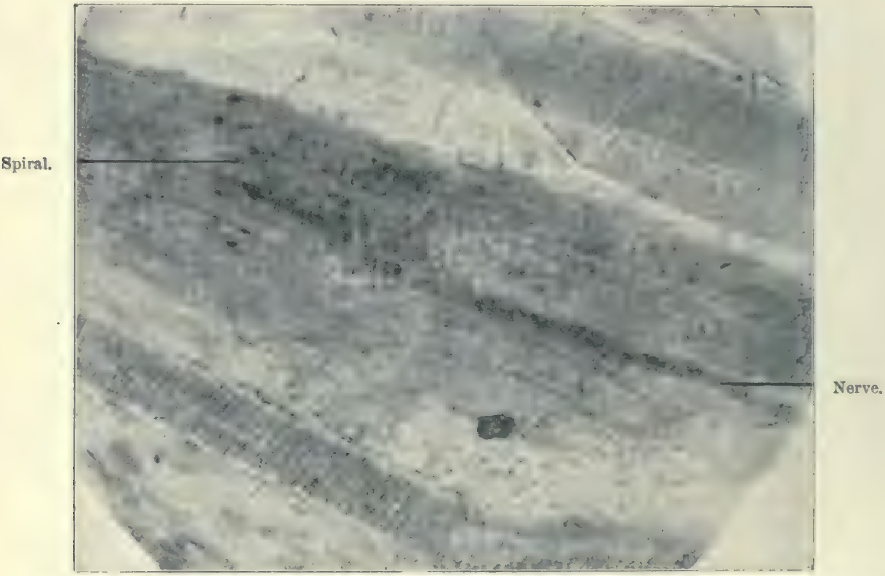


FIG. 2.—The same spiral nerve termination under higher magnification. The nerve can be seen passing from the left, it divides into two, the lower branch makes a double curve and passes to the lower spiral, the upper branch passes direct to the upper spiral. Between the coils of the spiral the outline of some of the large intra-muscular cells can be seen, and to the left the spiral can be seen passing on to the striated portion of the muscle fibre. Magnified 230 diameters.

spiral (plate I., fig. 5). Within a spindle two, sometimes three, of these spirals are to be seen, all presenting much the same appearance, they are always situated near the equatorial region, and lie parallel to one another (plate I., fig. 1 and plate II.). The spiral is sometimes double, the spirals running in the same direction, the coils lying parallel to one another and giving rise to " latticework " appearance (plate III., fig. 2).

(b) *At various times after Section of the Nerve.*

Twenty-four hours after Section of the Nerve.—The spindle itself presents no change, the nerve passing to it appears unaltered and the muscle fibres present no change. Certain changes are, however, already present in the spiral nerve termination, the spiral has become interrupted at intervals, it has lost its clear and distinct outline. The large intra-fusal cells remain unaltered (plate I., fig. 6).

Forty-eight hours after Section of Nerve.—The muscle spindle presents no change, the spiral has now, however, completely disappeared and in its place are seen oval and elongated cells with darkly staining borders and granular contents, but, containing no nucleus. The large intra-muscular cells are unaltered except for a slightly granular appearance (plate I., fig. 7).

Seventy-two hours after Section of the Nerve.—The spindle presents no change, the intra-muscular cells have become more granular and are less regular in shape and size, the oval and elongated cells do not seem to have altered much in appearance (plate I., fig. 8).

Ninety-six hours after Section of the Nerve.—The changes have become rather more marked, the large intra-muscular cells are more granular in appearance and more irregular in shape, some of them now seem to poseess a nucleus, the oval granular cells appear rather more numerous (plate I., fig. 9).

One hundred and twenty hours after Section of the Nerve.—No marked alteration is to be noticed from the previous specimen, except that possibly the oval granular cells are fewer in number (plate I., fig. 10).

Seven days after Section of the Nerve.—The spindle is still easily teased out and in shape does not appear abnormal. The oval granular cells seen in the early degeneration have now become fewer in number although some still remain. The intra-muscular cells have become somewhat shrunken in size, are irregular and granular in appearance. The nerve passing to this portion of the spindle can still be easily traced, but the staining is irregular, certain parts staining very darkly, other parts being unstained, the nerve seems to be broken up into elongated granular segments. The stiration of the muscle is well preserved (plate I., fig. 11).

Fourteen days after Section of the Nerve.—The spindle is easily recognised in the muscle although the guiding nerve fibres are no longer so distinct and there is a general increase of small round cells. The intra-muscular cells within the spindle are now very irregular in arrangement and shape, such as are seen are markedly granular, and form no distinct body within the muscle fibre (plate I., fig. 12).

II. The muscle spindle and the nerve termination as studied by—

(1) *Marchi's method.*—(*a*) *Under normal conditions.*

The normal muscle spindle stains a uniform brown colour by this method, the various parts of the spindle are easily recognised, but there is no contrast between the various parts of the spindle.

(*b*) *At various times after the Section of the Nerve.*

In the first seventy-two hours no change can be seen in the spindle and the finer nerve fibres.

In ninety-six hours although no obvious change is to be noted, the nerve fibres stain rather darker than normal.

In one hundred and twenty hours no further change is apparent.

At the seventh day after section of the nerve there is a large increase of fat around the spindle, and within the spindle fine, fat globules can be seen, but there is no obvious fatty change within the intra-muscular cells.

PLATE III.

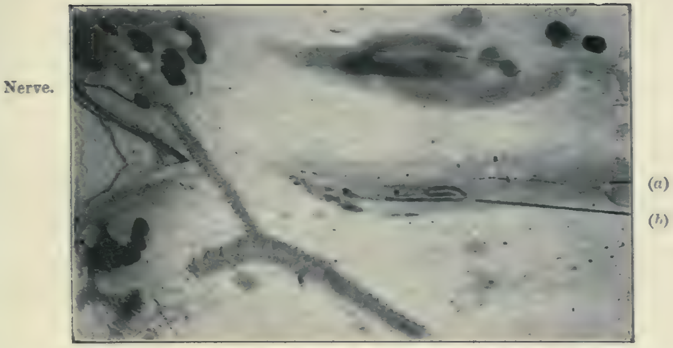


FIG. 1.—Photograph of longitudinal section of a normal muscle spindle stained by Marchi-Pal method. The nerve is seen passing to the spindle on the left. Owing to the spindle not being cut exactly in its longitudinal axis, an interruption of the nerve occurs. The nerve is again seen in the centre of the field, and dividing, sends one branch (*a*)—a nerve of small calibre—to the pole of the spindle: another thicker branch (*b*) passes to the equatorial region, where it makes a double turn and again divides. Under the microscope the intra-muscular cells can be seen just to the right of the first bend, and the striation of the muscle fibre is interrupted thereby. The spiral termination cannot be seen by this method.

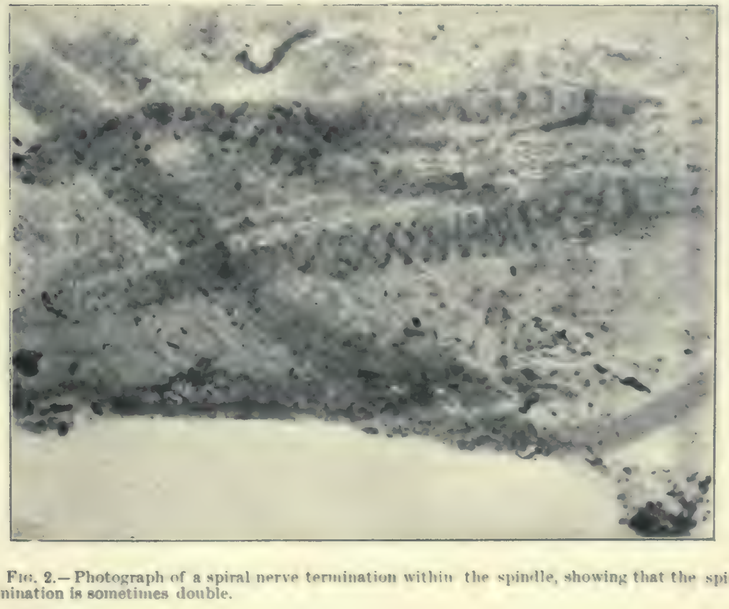


FIG. 2.—Photograph of a spiral nerve termination within the spindle, showing that the spiral termination is sometimes double.

Even at the fourteenth day after section of the nerve, although there are numerous fine fatty granules throughout the spindle, yet the changes shown by this method are not very striking.

(2) *Pal's method.*—(*a*) *Under normal conditions.*

In the normal muscle spindle the nerves passing to the spindle stain well, and the fibres can be traced into the spindle, the finer ones passing towards the poles, while the larger can be followed up to the point in the intra-fusal muscle fibre where the large cells above described exist, at this point the nerve seems to make a bend, sometimes double (plate III., fig. 1), and then may divide again on the surface of the muscle fibre (plate III., fig. 1). The spiral termination cannot be traced by this method, though there is possibly slight indication of it. The intra-muscular cells give to the muscle fibre in which they lie a granular appearance and do not appear so large as by Sihler's method.

At various times after Section of the Nerve.

During the first forty-eight hours after section no obvious change can be noted in the spindle. Seventy-two hours after section the nerve appears to be broken up into short segments; it stains, however, fairly well.

One hundred and twenty hours after section the breaking up of the fibres is rather more marked and the finer fibres stain less well.

Seven days after section the nerve has to a large extent lost its capacity of staining by Pal's method, the nerve stains in parts, the muscle fibre preserves its striation and the intra-muscular cells are not obviously altered.

Fourteen days after section the nerve stains very slightly by Pal's method. At certain points within the spindle there are small areas of black granules giving the appearance of some fatty degeneration, but the granules are not arranged in rows and do not give the usual appearance of degenerated nerve fibres.

By both the above methods some change is shown in the nerve fibre between the fifth and the seventh day after section of the nerve, but so far as the fine nerve fibres are concerned, the changes are by no means definite enough to be of much service.

At no time do the intra-muscular cells show any fatty change either by the Marchi or Marchi-Pal method.

III. The conditions of the nerve within the muscle, at the entrance into the muscle, and near its origin from the cord at various times after section of the nerve.

(*a*) *Sihler's Method (the following applies to the intra-muscular nerves).*

The primitive sheath of the normal nerve fibre is stained, the medullary sheath is unstained, or only slightly stained, and the axis-cylinder is as a rule somewhat slightly stained, the depth of staining being in part due to the penetration of the stain, there are some darkly stained nuclei lying in or below the primitive sheath. There is an absence of all granular appearance of the axis-cylinder. The nodes of Ranvier are best seen on the large nerve fibres.

Twenty-four hours after Section of the Nerve.—The axis-cylinders begin to have a slightly granular appearance, and are interrupted in places.

Forty-eight hours after Section of the Nerve.—The axis-cylinders have a more granular appearance.

Seventy-two hours after Section of the Nerve.—The axis-cylinders tend to become thin in parts, and are broken up in segments, and are granular in appearance.

Ninety-six hours after Section of the Nerve.—The axis-cylinders now stain in patches, certain portions being more darkly stained than normal, while other parts are but slightly stained, the segmentation is marked, and the nodes of Ranvier darkly stained.

One hundred and twenty hours after Section of the Nerve.—There is no marked change from the foregoing observation.

Seven days after Section of the Nerve.—The segmentation of the fibres is much more marked, and the finer fibres are broken up into elongated shaped pieces, which are extremely granular.

Fourteen days after Section of the Nerve.—The axis-cylinders have in places become very thin, with granular masses at intervals, the general appearance of the nerve is that it stains less than the normal nerve.

(*b*) *Marchi's Method.*

The normal nerve fibre stains a light brown colour, and at a few points some darker pigmentation of the nerve occurs, situated usually on the outer side of the medullary sheath and not at the nodes of Ranvier.

Twenty-four hours after Section of the Nerve.—No change can be noted.

Forty-eight hours after Section of the Nerve.—The medullated sheath of the nerve is possibly in parts more darkly stained than normal, and this is more obvious in the upper two portions of the nerve than in the nerve within the muscle.

Seventy-two hours after Section of the Nerve.—The pigmentary changes are now seen in the intra-muscular nerve fibres, and appear to be less in the middle segment than higher up.

Ninety-six hours after Section of the Nerve.—The pigmentation is more marked at all levels examined, but it has not the characteristic appearance of fatty degeneration.

One hundred and twenty hours after Section of the Nerve.—No further change is to be noted.

Seven days after Section of the Nerve.—The degenerative changes have become marked, though not extensive, the changes appear to be about equally marked in the whole course of the nerve, and not more marked peripherally than centrally.

Fourteen days after Section of the Nerve.—The degeneration of the nerve is now well marked in the whole course.

(*c*) *Pal's Method.*

The normal nerve fibres stain well by this method, the medullary sheath staining darkly with a regular outline, leaving the central portion of the fibre clear.

Twenty-four hours after Section of the Nerve.—No change is to be noted.

Forty-eight hours after Section of the Nerve.—The nerve shows a somewhat beaded appearance, the fine intramuscular fibres do not show this change so markedly as the larger fibres.

Seventy-two hours after Section of the Nerve.—The beaded appearance of the nerve is rather more obvious.

Ninety-six hours after Section of the Nerve.—The nerve begins to stain irregularly, and the segmentation of the fibre is more marked.

One hundred and twenty hours after Section of the Nerve.—The nerve presents the same appearance as above described.

Seven days after Section of the Nerve.—The nerve stains very irregularly, the medullary sheath is broken up into a number of fine, darkly staining granules, and in many parts the sheath no longer stains.

Fourteen days after Section of the Nerve.—The nerve has still further lost its capacity of staining by this method, and the degeneration is equally marked in the intra-muscular nerves, in the nerves near their entrance into the muscle, and in the nerve near its origin.

With regard to the above two methods, although certain changes in the nerve may be noted as early as the seventy-second hour after section of the nerve, yet the changes do not become definite and reliable till between the fifth and seventh day after section of the nerve, and it cannot be said that changes make their appearance in the peripheral portion of the nerve before the more centrally situated portion of the nerve.

(4) Summary of Results Obtained.

(*a*) The examination of the muscle spindle in dogs has shown the existence of a spiral form of nerve termination surrounding a fine muscle fibre, in the centre of which there exists large, clear, non-nucleated cells. These cells are shown to tail off at either end into smaller cells, which lie in the centre of the muscle fibre.

This spiral is shown to be in connection with a large nerve fibre.

(*b*) It is also shown that twenty-four hours after section of the nerve changes are present in this spiral termination, and become marked in forty-eight hours, such changes consisting of breaking up of the spiral, the appearance of elongated and oval granular cells. Further changes take place in the intra-muscular cells, and also in the axis-cylinders of the intra-muscular nerves at an early period after section of the nerve.

(*c*) It is also shown by Marchi and Pal methods that degeneration of the medullated sheath of the nerve does not become apparent until between the fifth and seventh day after section of the nerve, and then is present in the whole course of the nerves at the same time.

(5) COMPARISON OF RESULTS WITH THOSE OF OTHER OBSERVERS.

In 1888 Cattaneo[1] showed by means of a gold chloride method of staining in rabbits and guinea-pigs that changes were present in the nerve termination in the musculo-tendon organ twenty hours after section of the nerve, such changes disappearing after the third to the fourth day after section of the nerve.

Although he describes the muscle spindle, he does not recognise its close relationship to the musculo-tendon organ.

He further adds that which I think is borne out by the present series of experiments, viz., that the degeneration of the medullated portion of the nerve is slow compared to that of the non-medullated part.

In 1893 Ruffini,[4] using the gold chloride method, described in cats an annulo-spiral termination within a muscle spindle, and he gives a diagram of the appearance, which certainly closely resembles that which I have described in this paper, but he does not describe or figure the large intra-muscular cells nor the interruption of the striation of the muscle fibre which occurs at this point.

In a recent paper in BRAIN[5] he refers to three forms of the nerve ending within the muscle-spindle, and the one described in this paper undoubtedly corresponds with his "primary form of ending," as it is the one connected with the large nerve fibre. In a still more recent publication in the *Journal of Physiology* he gives a detailed description and diagrams of the three forms of nerve ending, the primary spiral termination, the secondary "flowered ending," and the tertiary "plate" ending which is not motor.

In 1896[6] Weiss and Dutil, using gold chloride and other methods described in guinea-pigs, rabbits, and cats, the large intra-muscular cells and the nerve as terminating between these cells, these they figure in their plates, but they do not figure any spiral nerve termination round these nuclei.

Kerschner[3] also showed before the Anatomical Society in Vienna a specimen demonstrating a form of nerve termination winding round the muscle fibre within the muscle-spindle.

In 1890[2] Dogiel, using the methylene blue method, describes a spiral in frogs in connection with a nerve fibre of large size, but does not figure it.

Quite recently, in March of the year 1898, Huber and De Witt figure a spiral nerve termination obtained from amphibia, reptiles, birds, and the mammals.

(6) CONCLUSIONS.

The first object of the research was to ascertain whether degeneration of that part of the neuron most remote from the cell took place before other parts of it were similarly affected, and in regard to this first point, it has been shown that after section degeneration of the medullated sheath of the nerve takes place in the whole course of the nerve at the same time, and that the centrally situated portions of the nerves suffer as soon as the peripheral. With regard to the axis-cylinder, it has been shown that changes take place in the terminal portion within twenty-four hours after section of the nerve, but it has not been proved that similar changes do not exist in the axis-cylinder near the cell, the methods employed failed to demonstrate this point.

The second object of this research was to reproduce, if possible, in animals the changes which I have shown to be present in the terminal end organ in the case of tabes dorsalis in man, and here the experiments failed to show by the Marchi method those fatty changes in the intra-muscular cells which were found in the above case.

REFERENCES.

(1) Cattaneo. *Archiv. Ital. de Biologie*, tom. x., p. 337, 1888.
(2) Dogiel. *Archiv. für Mikroscop. Anat.*, bd. xxxv., s. 305, 1890.
(3) Kerschner. "Verhand. der Anat. Gesel. fürdie sechste Versammlung," s. 85. Wein, 1892.
(4) Ruffini. *Archiv. Ital. de Biologie*, tom. xviii., p. 106, 1893.
(5) Ruffini. "Brain," 1897.
(6) Weiss and Dutil. *Archiv. de Physiologie*, tom. viii., p. 363, 1896.
(7) Batten. "Brain," p. 138, 1897.
(8) Huber and De Witt. *Journal of Comparative Neurology*, vol. vii., p. 169, 1898.
(9) Ruffini. "Journal of Physiology," vol. xxiii., p. 190, 1898.

DESCRIPTION OF PLATE I.

Fig. 1.—Drawing of spiral nerve termination within normal muscle spindle made under the camera lucida. The relative positions of the spirals and the intra-muscular cells is well seen. Two fine nerve fibres are seen below the spirals.

Fig. 2.—The same spiral under higher magnification, showing junction of nerve with spiral, and the tailing off of the intra-muscular cells.

Fig. 3.—Drawing from another spindle to indicate the position of the spiral nerve terminations within the spindle.

Fig. 4.—Drawing of the same spindle to show the spiral nerve termination (A).

Fig. 5.—Drawing of the same spiral under higher magnification, showing the tailing off of the intra-muscular cells in both directions.

Fig. 6.—Drawing from part of an intra-fusal muscle fibre containing the large intra-muscular cells twenty-four hours after section of the nerve. The nerve is seen approaching from the left, it looses its myelin sheath and divides into two, one part going to the first coil of the spiral, the destination of the other is not apparent, the spiral is broken and irregular in outline, the intra-muscular cells are in this specimen indistinctly seen.

Fig. 7.—Drawing from part of an intra-fusal muscle fibre containing large cells forty-eight hours after section of the nerve, showing the disappearance of the spiral and the appearance of the large oval granular cells, the large intra-muscular cells remaining unaltered. The nerve can be seen passing from left to right and then bending round and terminating on the under side of the lower spiral.

Fig. 8.—The nerve termination seventy-two hours after section of the nerve. The intra-muscular cells have become slightly granular.

Fig. 9.—The nerve termination ninety-six hours after section of the nerve. The intra-muscular cells are more irregular in arrangement and somewhat granular in appearance, the oval granular cells are still present.

Fig. 10.—The nerve termination one hundred and twenty hours after section of the nerve. The condition does not differ from that given of the last specimen, except that there are fewer large oval cells. The drawing is made towards the tail of the intra-muscular cells.

Fig. 11.—The nerve termination seven days after section of the nerve. The large intra-muscular cells are somewhat shrunken, and the oval granular cells fewer in number. In the lower muscle fibre the tail of the intra-muscular cells is seen, the bulk of the cells lying more to the right.

Fig. 12.—The nerve termination fourteen days after section of the nerve. The termination has undergone very considerable alteration, so that it is not easily recognisable. The intra-muscular cells have become very irregular. All the above drawings were made under the camera lucida.

TABLE I.

Nerve and Nerve Termination within the Spindle.

	SIHLER'S METHOD.	MARCHI.	PAL'S METHOD.
DOG, I. Normal.	Spiral nerve termination. Intra-muscular cells.	Nerve termination not stained, intra-muscular cells give to muscle fibre a granular appearance.	Intra-fusal nerve stains well up to commencement of spiral nerve termination.
DOG, II. 24 hours after section of the nerve.	Spiral termination interrupted. Intra-muscular cells unaltered.	Unaltered.	Unaltered.
DOG, III. 48 hours after section of the nerve.	Spiral has disappeared. Large oval cells are present. Intra-muscular cell slightly granular.	Unaltered.	Unaltered.
DOG, IV. 72 hours after section of the nerve.	Large oval cells as above. Intra-muscular cells more granular and less regular in shape.	Unaltered.	The nerve now appears broken up into short segments, it stains, however, well.
DOG, V. 96 hours after section of the nerve.	Intra-muscular cells more granular and irregular in shape.	The nerve stain somewhat darker than normal.	Unaltered.
DOG, VI. 120 hours after section of the nerve.	Same as above. Oval granular cells fewer in number.	Unaltered.	The breaking up of the fibres is more marked, and the fine fibres stain less well.
DOG, VII. 7 days after section of the nerve.	Intra-muscular cells more shrunken.	Some fat globules seen within the spindle but degeneration not obvious. Intra-muscular cells show no fatty change.	The nerve has lost to a great extent its capacity of staining by Pal's method. No change seen in intra-muscular cells.
DOG, VIII. 14 days after section of the nerve.	Intra-muscular cells very irregular in arrangement, and those seen are very granular.	Fat seen within spindle but degeneration not striking. Intra-muscular cells show no fatty change.	The nerve stains very badly by Pal's method, no fatty change seen in the intra-muscular cells.

TABLE II.

	Condition of Nerve within Muscle.		*Condition of Nerve at entrance into Muscle.*		*Condition of Nerve near its Origin.*	
	MARCHI.	PAL.	MARCHI.	PAL.	MARCHI.	PAL.
DOG, I. Normal.	Nerve stained light brown. No dark pigmentation seen in specimen.	Well stained. Nerves have distinct regular outline.	Nerve light brown colour. Nodes of Ranvier distinct. Some few dark (almost black) patches in the course of some fibres.	Centre of fibre clear. Contour of fibre uniform. Well-stained sheath. No appearance of segmentation.	Nerve fibres brown colour. Nodes of Ranvier clear and unstained. A few dark patches in course of fibre.	Centre of fibre clear, well-stained sheath.
DOG, II. 24 hours.	No alteration.	No alteration.	No alteration.	No alteration.	No alteration.	No alteration.
DOG, III. 48 hours.	No alteration.	No alteration.	Darkly stained patches more numerous. Not definitely fatty.	Nerve begins to have beaded appearance.	Pigmental patches more numerous.	Nerve has a beaded appearance.
DOG, IV. 72 hours.	Some fibres now appear pigmented (not fatty).	Beaded appearance probably present. (Specimen unsatisfactory.)	Darkly stained patches less numerous. Contour of fibres is irregular. Beaded appearance of fibre and numerous dark dots present.	Beaded appearance of nerve fibre, stain irregular.	Pigmented patches more numerous, and have a dark granular appearance, and are imposed in the sheath of the nerve.	Nerve appears beaded.
DOG, V. 96 hours.	Same as above.	Nerve begins to stain irregularly. The myeline sheath begins to break up.	Numerous dark dots present. Typical degeneration not yet present.	Irregular staining more marked.	Black dots present. Typical degeneration not present.	Stain irregularly.
DOG, VI. 120 hours.	Same as above.	Same as above.	No further change.	No further change.	No further change.	No further change.
DOG, VII. 7 days.	Degeneration now obvious, affecting the smaller fibres most.	Nerve stains badly. Sheath of nerve broken up into small rounded dots.	Degeneration now marked but not universal.	Nerve broken up. Numerous fine darkly stained granules.	Degeneration marked, but apparently not so extensive as low down in the nerve.	Nerve is now broken up into segments. Numerous fine dark stained granules.
DOG, VIII. 14 days.	Degeneration marked.	Nerve stains badly, most of the fibre unstained.	Degeneration is very marked.	Degeneration very marked, the section shows a good deal more darkly stained granules than the Marchi specimen.	Degeneration is very marked.	Degeneration is very marked.

NERVOUS SYPHILIS;

With a Critical Digest.

BY W. JULIUS MICKLE, M.D., F.R.C.P.LOND.

(*Continued from* BRAIN, *Summer Number*, 1898, *p.* 249.)

CLINICAL, AND OTHER FORMS AND CONDITIONS OF NERVOUS SYPHILIS.

IN my previous articles in BRAIN[1] on this subject the material was divided in a definite way, merely as a convenient method of dealing with certain particular material relating to part of the field of study presented by nervous syphilis. As was explicitly stated in the remarks introductory to those articles, "a convenient division of the material in hand is into six Parts." Never for a moment was it intended to serve as a formal classification of nervous syphilis. But it has been taken so by Dr. C. K. Mills, and, under this misunderstanding, he makes criticism which would justly apply had an attempt been made to offer as a formal classification of the whole subject what was merely a convenient division of material bearing on parts of it. The division will still be convenient to continue, and thus bring much of the following into line with the preceding contribution.

VII.—SOME FORMS OF EXTENSIVE OR MULTIPLE SYPHILITIC DISEASE OF NERVOUS SYSTEM; CHIEFLY CEREBRO-SPINAL.[2]

There are some differences between the conditions which, when syphilis attacks the cerebro-spinal nervous system, predispose to its incidence on the brain or on the cord. These we need not consider here.

A recent article by Kuh offers a separation into forms of luetic spinal disease, which is a cross division, partly pathological, partly clinical; namely, into syphilitic spinal meningitis, syphilitic spinal paralysis, syphilitic transverse myelitis, syphilitic acute myelitis. And as regards the brain, it divides into syphilitic meningitis of the base of brain, that of its convexity, syphilitic arteritis, gumma.

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[1] BRAIN, Spring Number, and Summer Number, 1895.

[2] See also Part I., BRAIN, Spring Number, 1895, p. 102, *et seq*.

In relation to some forms of severe or extensive or multiple syphilitic disease of central nervous system, one must bear in mind that syphilis may engender widely-spread conditions of arteritis, or of sclerosis, or of degeneration. These last are in some cases *direct ;* in other cases they are *indirect and secondary* degenerations, either of (*a*) Wallerian type, or of (*b*) retrograde type. Several grave para-syphilitic affections also have an extensive or a multiple distribution.

In reference to mental disease, the frequency of states of mental depression of syphilitic origin is noteworthy. Noteworthy, also, is the variety of these states of depression, and the numerous different conditions of syphilitic change on which they may be founded. The general history; the evidence of other symptoms and physical signs clearly due to syphilis; the random association and succession of symptoms; the bazarrerie and multiplicity of the clinical phenomena; the peculiarities of some of them—will be of greater help, than any purely mental symptom-group, towards the indication of a syphilitic origin. On the mental-symptom side the exceptions will be more numerous than those conforming to the proffered rules. In this relation, we may mention a recent attempt by Spitzka to detail the differential diagnosis between "genuine melancholia" and a somewhat similar condition observed in the initial period of syphilitic dementia, as follows :—The sadness of the true melancholiac is primary ; the other is depressed because of inability to attend to business, and suspicion that the friends realise this. The former suffers from insomnia ; but the latter sleeps well and often over-much. The true melancholiac neglects all his dress; the other only neglects part of *his*, being very particular about other parts. The former shrinks from company ; the latter is irritable, and his symptoms are very variable. The syphilitic finally passes into a condition in which are slight but characteristic changes in the medulla oblongata.

A well-reported case of luetic meningo-myelo-encephalitis was published by Alzheimer. In it was syphilitic meningo-myelitis (distinguished from gummatous disease and from the acute mylitides after syphilis), together with a corresponding condition of brain meninges and cortex, chiefly affecting the convexity.

During life, the case had been diagnosed as general paralysis *plus* spinal-cord syphilis. The existence of these, rather than of ordinary brain-syphilis, seemed to be supported by the severe dementia, confusion, exalted ideas, rapid course; and by the absence of implication of other cranial nerves than the optic, and absence of the coming and going and inconstancy of the symptoms, so frequent in brain-syphilis.

The basal meningitis was slight, whereas the convexity was considerably affected, and the meningitis there, in a markedly irregular, unequal way, circumscribed in its more severe incidence.

Comparing the vascular change in general paralysis and in this case: in the former are numerous nuclei of most different form, in extremely various stages of destruction, and losing organisation; in the latter much more numerous nuclei, quite corresponding to those of the spinal vessels, all alike, and none degenerate: in the former a chronic process with exacerbations; in the latter a simultaneously occurring infiltration.

In my previous article it was mentioned that spinal and cerebro-spinal cases sometimes throw light upon obscure or contested points concerning the more local or systematic morbid processes connected with syphilis. As a further exemplar may be cited a case of cerebro-spinal syphilis, reported by Cassirer, to which, indeed, recurrence will be made in a subsequent section.

Clinical Summary.—Headache; right hemiplegia, becoming spastic with increased tendon-reflexes; also less increased tendon-reflexes left side; articulatory disorder of speech; hallucinatory excitement; general sclerotic state of arteries; unequal pupils; right reflex iridoplegia; transitory diplopia; left ptosis; right facial paresis; transitory attack simulating acute meningitis.

Necropsy.—Pachymeningitis in pontine and cerebellar regions.

Lepto-meningitis invading nervous substance in all parts examined.

Gummata of crura cerebri.

Degeneration of intra-medullary part of 4th left nerve-root.

Degeneration of intra-medullary part of 5th right nerve-root.

Secondary degeneration of pyramidal lateral tracts.

Degeneration of posterior spinal root-zone in left lower cervical region.

Chronic arteriitis—affecting all coats—in all regions examined.

Although the vascular and meningeal changes were not absolutely specific, they make a syphilitic basis probable; especially the alliance of endo-, meso- and peri-arteriitis; and the invasion of nervous substance by meningitic processes. Also the existence of early gummata tells that way. Also the variety of nervous lesions found; and perhaps the liver-change.

The hemiplegia and descending degeneration were from destruction of left pyramidal fibres in the foot of the cerebral peduncle. As to the severe degeneration of intra-medullary part of left 4th cranial nerve, other cases have been observed, and there is a variety of possible causative conditions; and in syphilitic infiltration of the cranial nerves at the base, the 4th is never affected alone.

Degeneration of spinal trigeminus nerve root was in this case unilateral; in tabes it is usually bilateral.

The degeneration of posterior root-zone in this case was most interesting, and will be taken up, later on, in relation to tabes and syphilis. See Section X.

In almost any series of cases under the present heading many suggestive particulars occur; *e.g.*, in the first at hand, by Stieglitz, is a case resembling an early stage of general paralysis, but diagnosed to be local syphilitic meningo-encephalitis. Another beginning with general malaise, fatigue, insomnia, and slight numbness of left hand; later on, spells of vomiting without nausea; hiccough, constant and persisting day and night; and, later still, staggering towards the left side:—

A case of cephalalgia of fifteen years' standing, cured by K.I.

One with, *inter alia*, temporary verbal amnesia and transient alexia; and, in connection with the verbal amnesia, "sensory focal discharges."

Early nervous symptoms, occurring within the first few months after infection, have been reported in many cases; as by Hutchinson, Gowers, Brasch, Lamy, myself; and by those others I cited in the "Spring" and "Summer" Numbers of Brain for 1895.

Much might be added here to what has been stated on the relations of syphilis and general paralysis, but these will be dealt with in a subsequent contribution.[1]

A bewildering variety of extensive lesions, luetic in origin, were reported in a case by Dr. H. M. Thomas. Syphilitic orchitis, gummatous endo-arteriitis of cerebral arteries; gummata of five different cranial nerves, of left crus cerebri, and in the brain.

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[1] Since this was written, an article by Dr. W. R. Dawson deals with syphilis and insanity (*Journ. Ment. Sci.*, April, 1898).

Meningitis of cord. Poliomyelitis of its lumbar region; and, here, the anterior grey horns, the site of capillary hæmorrhages, in parts disintegrated and the grey matter extensively destroyed, the blood-vessels, here, much diseased; the ganglion cells mostly disappeared; spinal pia intensely infiltrated with small, round cells; hyaline degeneration of many small vessels of pia and cord; in parts, typical endo-arteriitis obliterans.

Differential Diagnosis between three forms, (a) Arterial, (b) Meningeal, and (c) Gummatous, Syphilis of the Brain.

On this subject an article[1] is published by Teissier and Roux, and is summarised here.

Although the symptoms dependent upon these three kinds of lesion depend more upon their localisation than upon their anatomical nature, nevertheless this last can sometimes be arrived at by semeiological study.

I. In the first, or *Arterial Syphilis*, the phenomena of *failure* ("deficit") dominate over those of *irritation*. Let us examine the symptoms referable to (*A*) *Motion;* (*B*) *Sensibility;* (*C*) *Intellect.*

(*A*) *Motor Symptoms.*—By far most often is a flaccid paralysis with abolished reflexes: hemiplegic, monoplegic. Rarely is there spasmodic paralysis, with rigidity, contractures, exaggerated reflexes [except as a quite secondary condition.—W. J. M.].

Partial "epilepsy" is less frequent than in the other two forms; and is apt to be less localised, less monoplegic. Some form of intoxication is usually a secondary co-operating excitant of convulsion: to arterial ischæmia, intoxication is superadded.

(*B*) *Disorders and Loss of Sensibility.*—Headache, usually affecting one side of head, not aroused by pressure or percussion of cranium, often deep-seated.

Sometimes very transitory numbness or formication.

Objective sensory symptoms usually absent, except when a certain part of internal capsule is lesed.

Special senses usually unaffected. No hallucinations.

Ocular paralyses rare.

(*C*) *Intellectual Symptoms.*—Most often, these consist of a progressive weakening of mind generally, without active delirium. Varieties of aphasia are frequent.

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[1] *Archives de Neurologie*, January, 1898.

As regards *the evolution and groupings of the symptoms*, there are two periods: that of *curable* symptoms and that of *incurable*.

In the curable period :—

(*a*) *If the arteritiis is* OBLITERATIVE, there come cerebral ischæmia and intermittent cerebral "claudication." Then overwork or any kind of intoxication easily makes this brain give way.

The symptomatic forms in the curable period are chiefly three :—

(1) *Paralytic :* paralyses, flaccid, sudden, local or hemiplegic, with abolished reflexes and without sensory impairment.

(2) *Aphasic ;* transitory, intermittent.

(3) *Intellectual ;* mind depressed, failing, weak, demented.

(*b*) *If the arteriitis is* DILATING : to the above symptom-groups are added very various symptoms due to compression of this or that part of intra-cranial contents by the aneurysmally dilated vessels.

In the incurable period :—

May be thrombosis, brain-softening, cerebral hæmorrhage: a fatal apoplectic form may arise either from sudden obliteration or from vessel-rupture.

II.—MENINGEAL SYPHILIS OF BRAIN. SYPHILITIC MENINGITIS.

Purely meningeal cases, only, are included here; and three varieties of lesion :—(1) Gummy infiltration; (2) disseminated miliary gummata; (3) inflammatory or sclerous meningitis, "without other specific character than its ætiology and the habitual efficacy of treatment."

In meningeal syphilis, the phenomena of irritation predominate over those of failure and defect.

(*A*) *Motor Troubles.*

Epileptiform attacks; particularly frequent in this form. Paralyses or pareses; which usually are accompanied from the beginning by spasmodic phenomena, *i.e.*, by muscular rigidities, contractures, exaggerated reflexes, often cloni; paresis and rigidity being the most frequent association. The paralyses are more diffuse and less complete than in arterial syphilis, as a rule; and often with them are involuntary jerks and twitches.

(*B*) *Sensory Disorders.*

Much more frequent than in *arterial* syphilis, and far more permanent.

Headache scarcely ever fails; may be generalised or localised; most often is associated with superficial hyperæsthesia; is brought out or aggravated by pressure or percussion of cranium.

Very lively *pains* may affect *limbs, trunk or viscera*, and be of central origin (cortical irritation ?).

Objectively, may be found zones of *hyperæsthesia, paræsthesia, anæsthesia.*

The above disorders of sensibility are more permanent than the similar symptoms from arterial syphilis producing transitory ischæmias. Relatively permanent and intense, they often are the source of illusions, hallucinations, and delirium. *Sensorial* symptoms are frequent *e.g.*, visual. Frequent, also, are the *oculo-motor.*

Optic neuritis is frequent; the inflammation often very marked. Sometimes *primary optic nerve atrophy* occurs.

(*C*) *Mental Troubles.*

Aphasias; less complete, but more permanent, than in arterial syphilis. Exceptions occur in both forms.

Delirium.—Active delirium; agitation perhaps extreme, and giving a likeness to acute mania. Hallucinations are frequent, and become sources of delirious interpretations, of transitory notions of persecution, of having undergone operation, of being beheaded or dead.

Early trophic symptoms may arise and be of gastric origin; or extreme denutrition may be of cortical origin and remind one of severe general paralysis.

Apoplectiform stroke, with specific cortical encephalitis.

Fever, relatively frequent in meningeal syphilis; somewhat rare in arterial brain syphilis; exceptional in brain-gumma.

EVOLUTION AND SYMPTOMATIC GROUPINGS OF MENINGEAL SYPHILIS.

An acute form and a chronic exist; and of each a basal and convexal variety.

(*A*) Acute syphilitic meningitis. Rare. Begins by very intense headache, repeated vomiting, raised temperature. If the meningitis is *basal*, later on are added vertigo, mental symptoms, compression of cranial nerves at base, bulbar symptoms, polyuria; deep depression; final coma. But if the acute meningitis is of the *convexity* the supervenient symptoms are noisy delirium, hallucinations, repeated convulsions, paralyses, final coma. Specific treatment is useless.

(*B*) Chronic syphilitic meningitis. Frequent. A curable stage precedes an incurable. In the curable stage are slight and fugitive symptoms, convulsive, paralytic, or aphasic.

Much the more frequent variety of the *chronic meningitis is that of the Base.* At the brain-base, the compression exercised on cranial nerves and on crura cerebri or pyramids yields all the varieties of *alternate hemiplegia.* Frequent are polyuria and polydipsia, depression, somnolence, coma, bulbar symptoms.

With chronic meningitis of the brain-convexity, goes excitation of motor, sensory, and intellectual functions. Strokes are frequent; coma is rare.

III.—Gummatous Brain Syphilis.

Included here are only the gummata developing and remaining within the cerebral substance.

As to the *general characters* of the symptoms of brain-gumma; there is union of both irritative symptoms and symptoms of defect or failure (the positive and negative). These indications of defect or failure vary greatly with the site of the syphilomata, and differ much according as a given neoplasm affects the lines, pathways, or zones, of *association;* or those of *projection.* In the latter case, the symptoms are much more striking and more easily observed. Very varied, also, are the irritative symptoms; motor, sensory, intellectual; and clinically and pathogenically they are those found in brain tumours generally.

As to *pathogenesis;* they may arise from (*a*) extension of the lesions progressively; or from (*b*) congestion around these; from (*c*) distal action or effect; from (*d*) vascular compression; or from (*e*) increased intra-cranial tension.

Great importance is attributed by Teissier and Roux to toxic and auto-toxic agencies—endogenous and exogenous—in the production of symptoms with brain-gumma; also of the congestions often occurring around or with it; the softening about it; the accompanying œdema of optic disc; the usefulness of intestinal and other antiseptics in sometimes clearing up inter-current symptoms.

Ophthalmoscopically; choked disc (œdema, stasis, hæmorrhage) is the most frequent abnormality, although a degree of optic neuritis is sometimes observed.

In its evolution, brain-gumma exhibits all the symptoms of brain tumours.

It proceeds by successive *exacerbations*, with periods of *remission*.

A basis of defect is always present; the "irritative" phenomena supervene.

The *course* is affected by the circumstance whether the gumma is diffusely infiltrating, or, on the other hand, is encapsuled; in the latter case the liability to auto-intoxication and to "irritative" phenomena is less.

In fine:—Whereas in arterial and in meningeal syphilis we *first* diagnose brain syphilis, *then* the variety. On the contrary, in brain-gumma we *first* diagnose brain-tumour, *then* its luetic origin and nature.

In brain-gumma, spontaneous remissions of the symptoms may occur, so may intermissions, even of long duration; and under anti-luetic treatment there may be complete and lasting cure. Rarely big, these syphilomata are often plural, or even numerous.[1]

In relation to the foregoing it is well to repeat that these three several forms of brain-syphilis are more or less mixed, as a rule. *E.g.*, with brain-gumma there is usually some syphilitic meningitis, or arteritis, or both. It is not very frequent, therefore, to find the clinical aspect of the cases so sharply and distinctively defined as represented here.

VIII.—Some Aspects of Congenital or Hereditary Syphilis.

Some aspects of congenital or hereditary syphilis formed the subject of the *second part* of my previous articles in this Journal for 1895, p. 130.

In a previous section of the present communication, those of the para-syphilitic affections which are due to *hereditary* syphilis received some attention. Therefore a brief recapitulation will suffice here.

These para-syphilitic affections from the hereditary form of syphilis are said to be chiefly as follows:—

Fœtal cachexy, involving more or less inaptitude for life.

Various congenital malformations.

Certain general or partial dystrophies by retardation or arrest of general or local development.

Morbid tendencies to disease of nervous system.

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[1] See also Oppenheim. *Specielle Path. und Therapie.*, edited by Nothnagel.

Morbid alterations, such as rickets, hydrocephaly, meningitis, congenital spasmodic tabes, infantile or juvenile tabes dorsalis and general paralysis.

And perhaps such dynamic changes as are involved in some examples of neurasthenia, hysteria, or epilepsy, in early life.

To the faults and errors of this classification, reference has already been made.

Hoffmann (of Heidelberg) describes the case of a lad, aged 14 years, with spastic spinal paralysis as part of an hereditary syphilitic affection of the central nervous system. The spastic symptoms began at the age of 12 years. The boy's mental development had been imperfect and tardy, and finally came to a standstill; and his physical growth was backward. These, and the spastic symptoms, and more or less complete internal ophthalmoplegia, were the main clinical features.

The author holds that the disease sprang from the hereditary syphilis. Moreover, that the cerebrum was lesed; the pupillary state dependent upon the same anatomical lesion as in tabes; the spastic condition due to spinal changes independent of the cerebral and therefore primary, or else secondary and dependent on a degeneration of pyramidal tract following brain lesion, although a third possibility was that pyramidal degeneration might be absent (as in a case by Zacher).

Besides the obvious developmental arrest, there was evidently an active process of disease, altering functions previously more or less normal, and producing spinal lesions which were probably of para-syphilitic nature. And Hoffmann inclines to believe that the spinal change was co-ordinate with, and not subordinate to, the brain change, and that syphilis played here much the same part as heredity does in many "system diseases" (as Friedreich's ataxy; Strümpell's hereditary spastic spinal paralysis) and that the lesion was not a genuine pure syphilitic one. He rejects the suggestions that the case was one of juvenile or developmental general paralysis, or that it was one of Little's disease (the regular basis of which, indeed, is a developmental defect rather than a degeneration). Besides the brain-change, a cord disease had arisen in a similar or like manner as in Minkowsky's case (acquired syphilis and primary lateral sclerosis of cord), or in Strümpell's hereditary spastic spinal paralysis, or in spastic general paralysis; and represented a case parallel to juvenile tabes dorsalis

Spiller cites the result of the examination by Gasne of the spinal cords of 36 fœtuses, of whom 26 were born of syphilitic parents:—"In four cases he found lesions of intense grade, identical with those seen in acquired syphilis, and in seven they were less important, but not doubtful. The meninges and vessels are most affected in the hereditary form, as in the acquired. He has examined critically all the cases of hereditary spinal syphilis reported, and concludes that the lesions are the same whether the disease is transmitted by the parents or acquired by the patient."

It is stated by Gilles de la Tourette that hereditary syphilis may attack the cord (1) at intra-uterine life; (2) during infancy or childhood; (3) in adolescence or even in more advanced life. The first stage chiefly consists of diffuse inflammations of the cord and its meninges, somewhat comparable to interstitial syphilitic hepatitis. After birth may come true sclerosis of cord; sometimes with sclerosis of brain also. In childhood and adolescence the cervical cord region seems specially to be affected, and the mid-brain. As age advances, infiltrations may occur, especially the gummatous, in interstitial tissue around vessels or in membranes; and endo-arteriitis or endo-phlebitis may appear.

Fournier has recently discussed the forensic question of the syphilitic infection of the nurse by the nurseling. In deciding on occurrence, or not, of this form of infection, several precautions are obviously necessary. The nurse must first be thoroughly examined, and evidence of past or present syphilitic relic or lesion, other than that alleged to be from the child, must be carefully sought for. Then the child must be examined, and the question decided whether it is syphilitic, and, if so, whether the syphilis is congenital or acquired—for the child may have been infected by the nurse, and not the nurse by the child. In establishing congenital syphilis, the absence of chancre must be ascertained. It is also necessary to know when the secondary symptoms became manifest. Appearance of "secondary symptoms" during the first two months of life denotes that the syphilis is congenital. "Suppose that a child contracts syphilis on the day of its birth. The incubation period of chancre is three weeks at least—say, twenty days. The second incubation period averages about forty-five days—say, forty days; $20+40=60$. Therefore the secondary symptoms of acquired syphilis cannot appear before sixty days (two months)." (*Lancet*, January 22, 1898.) All the usual signs of congenital syphilis must be sought for, and the presence or absence of each of them carefully verified.

It has been held by some that syphilis may descend to the third generation. On this subject Dr. G. Ogilvie[1] concludes that there is a reasonable probability that this descent of syphilis to the third generation may occur; although none of the alleged cases which have been published are entirely convincing on the point.

IX.—Spinal Syphilis.

The Third Part of my previous articles on Nervous Syphilis concerned "Syphilitic Spinal Disease generally, but particularly of Spinal Cord, with Especial Reference to Syphilitic Spinal Paralysis; and to Tabetiform Cases."

In the first instalment[2] of those articles, and especially in the second instalment,[3] the fact has been sufficiently emphasised that syphilic spinal cord disease sometimes occurs relatively soon after syphilitic infection. And we need only refer to the subject now in order to reiterate that, in some examples of the kind, not only is the cord's implication at an early date but the clinical effect also comes on very rapidly. This rapid development of the clinical phenomena may occur also in a late, or comparatively late, stage of syphilis.

Some examples of nervous syphilis were published by J. L. Steven as being "sudden" in clinical development. The development may be rapid; rarely, if ever, is it sudden, although I have seen a spinal case in which it was almost sudden, but complicated by the effects of a drunken debauch.

This rapidity of development is often dependent on vascular lesions of the cord: arterial trunks or venous ones may be grossly lesed, and sets of smaller blood-vessels may be much diseased or even occluded, and thus the blood-supply of parts of the cord may be cut off more or less; the resultant local anæmia may rapidly starve the part; or syphilitic inflammatory invasion, or compression by the crush of its products, may squeeze the life out of portions of the cord.

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[1] *Lancet*, January 15, 1898. See also Dr. J. A. Coutts *against*, and Mr. Greene *for*, the possibility of this descent. *Ibid.*, January 22, 1898. Also on this subject, and hereditary syphilis, Dr. Geo. Ogilvie, *Brit. Jour. Dermat.*, October, 1897.

[2] BRAIN, Spring Number, 1895, *passim*.

[3] BRAIN, Summer Number, 1895, p. 352, *et seq.*

Let us recall to mind how the nervous substance may be affected by syphilis either *directly* affecting nervous parenchyma; or *indirectly* affecting it by producing inflammatory, or other, changes in vascular, lymphatic or connective tissue elements, to which affections of the parenchyma may become secondary; and let us also recall how the vascular lesions of both small and big blood-vessels (as in spinal cord and meninges) are vastly important in leading to defective nutrition, or even death, of parts of the nervous parenchyma by mechanical effects of the infiltration, or by invading extension of the morbid process from the meninges.

Necessary, also, is it to distinguish spinal cord changes directly due to syphilitic infiltration from those which are secondary systemic degenerations, and, I may add, from those, also, which are primary degenerations dependent on syphilis, and the pathogenesis of which may not be always the same.

Lamy and Nonne accept as forms of spinal syphilis:—

Syphilitic spinal meningitis;

Specific meningo-myelitis;

Specific myelitis (lepto-myelitis; diffuse cell-infiltration of cord; miliary gummata of cord; specific disease of spinal blood-vessels).

In my former article it was stated that up to the time it was written (1894) the anatomical lesion in *Erb's syphilitic spinal paralysis* was inferential; that the symptoms were attributed to a partial transverse lesion of the dorsal portion of the spinal cord, a local myelitis; that the lesion was inferred to be in the lower half of the spinal cord and posterior half of its transverse section. I stated that "the transverse section of cord is only partly lesed; apparently the posterior parts of lateral columns are chiefly affected, the posterior columns less affected (the posterior root-zone of which is only slightly affected as a rule), the posterior horns are also lesed (vesical, rectal, and sensory defects).

"Instead of myelitis, may not the change, in some cases, be due to syphilitic spinal arterial alteration, leading to (gradual) occlusion, and this to slow ischæmia and softening of spinal cord locally, followed by regressive changes in the softened patch, and by systematic secondary degenerations?"

Erb held that the clinical condition must correspond to a fixed definite anatomical lesion. Moreover, that this is in the dorsal cord and of relatively small length in longitudinal section, and affecting only parts of the posterior half of the cross section of the cord, as a rule symmetrically on both sides; and can scarcely be taken as one of the known system diseases; is not a "combined system disease" in posterior and lateral columns, this being excluded by the clinical picture, and by the absence of upward extension. The possibility of implication of Goll's column and of cerebellar lateral tracts was admitted. He suggested that the origin of the lesion was partly a specific luetic infiltration of the cord; partly a spinal cord alteration (degeneration) proceeding from luetic arterial disease. By this last he explained the partly rapid, partly slow onset, the remissions in the course, and the usually imperfect success of treatment.

But Muchin held that Erb's syphilitic spinal paralysis may be due to systemic generation, an opinion differing very considerably from that of Erb himself. He quoted a case of Westphal's[1] as being much like Erb's type, with (in upper part of thoracic cord) a focal (?) lesion of crossed pyramidal and direct cerebellar tracts and posterior columns, and apparently *ascending* degeneration, from it, in posterior columns, and peripheral parts of lateral columns; and *descending* degeneration (below the lesion) in lateral columns. But this last was taken by Westphal to be primary; and the "focal" nature of the lesion appears to be doubtful.

In certain cases (of Erb's "s.s.p.") the degeneration of lateral and posterior columns has been said to be secondary to ordinary syphilitic myelitis or meningo-myelitis; and this has been held to tell against the view that in cases of syphilis the cord degeneration may be primary, and that syphilis may give rise to combined systemic disease of the cord (as in pellagra). Nevertheless, some recorded cases and some opinions are in favour of the view that syphilis may cause such primary degeneration and combined system disease of cord (Westphal, Muchin, Nonne *et al.*). And syphilis can give rise to degeneration of posterior root-zones in cord (Cassirer *et al.*).

Kuh still attempts to distinguish as separate diseases syphilitic spinal meningitis, syphilitic transverse myelitis, syphilitic acute myelitis, and Erb's syphilitic spinal paralysis; and to separate them from such conditions as primary lateral sclerosis and non-syphilitic transverse myelitis.

From cases recorded in medical literature he adduced five as, in his opinion, affording the morbid anatomy of Erb's syphilitic spinal paralysis. Nonne analyses these and accepts only one, and that one doubtfully. Having thus practically set aside the cases adduced, he proceeds to exhume two cases himself as affording the anatomical basis of the disease; one from Westphal,[1] one from Minkowsky.[2]

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[1] *Archiv für Psychiatrie*, viii. and ix. Bd.

In the former case were primary degeneration of pyramidal tracts, and direct cerebellar lateral tracts, and ring degeneration; and degeneration of Goll's columns in the cervical and dorsal regions; consequently, a combined system disease. The membranes were healthy; the vessels of the cord presented only a simple secondary thickening of their walls.

In the latter case were symmetrical primary lateral sclerosis; degeneration of pyramidal tracts and cerebellar lateral tracts. The cord-vessels were widened, their adventitia was thickened. Spinal meninges healthy.

Then Nonne brings forward two cases of Erb's syphilitic spinal paralysis observed by himself.

In Case I. were :—

Degeneration of Goll's columns (2nd cervical to 6th or 7th dorsal N.)

Degeneration of lower lumbar lateral pyramidal tract.

Dorsal to 2nd cervical, some degeneration of lateral pyramidal tract, cerebellar lateral tract, and border zone of anterior columns.

Primary degeneration and simple atrophy of nerve-fibres; secondary overgrowth of neuroglia; secondary simple thickening of vessel-walls.

In this case there was primary degeneration, and not proceeding from the vessel, nor from a chronic meningitis, nor from a primary disease of ganglion cells. And Goll's columns were most affected above; the pyramidal most below; in harmony with *primary* degeneration of Goll's tracts being *downward;* and *primary* degeneration of the pyramidal being *upward.*

A combined system disease, therefore, was the basis of Case I.

In Case II. were :—

(1) Chronic transverse dorsal myelitis, and its attendant ascending and descending secondary degenerations; namely, secondary ascending degeneration of Goll's columns above the lesion; secondary descending degeneration of lateral pyramidal tracts below the lesion.

(2) Primary disease of cerebellar lateral tracts and of pyramidal lateral tracts *above* the lesion.

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[1] Westphal, *Archiv für Psych.*, xv. Bd.

[2] Minkowsky, *Deutsch. Arch. f. Klin. Med.*, xxxiv. Bd.

If those (as Oppenheim) who take the disease to be a stage in ordinary transverse dorsal myelitis claim this case in support of their view, yet is there also a primary degenerative process in it, namely "(2)" above.

The case did not start in the blood-vessels.

Erb's *localisation* is substantially confirmed by Nonne's two cases. But the suppositions that a syphilitic disease of blood-vessels starts the lesion, and that perhaps also a specific luetic infiltration of spinal cord occurs therewith, are negatived so far as concerns these two cases.

In Eberle's case were ascending primary degeneration of pyramidal lateral tracts, also descending degeneration of Goll's, Burdach's, and the cerebellar lateral tracts.

Consequently, primary combined (several) system-disease existed.

Nonne further mentions that Trachtenberg held Erb's syphilitic spinal paralysis to be a toxic post-syphilitic system-disease, analogous to the system diseases of the spinal cord from ergot, or from lathyrus. And we have seen that Muchin deemed it to be, at least in some cases, a system-form of change, syphilitic in origin.

To sum up:—Thus in several cases at least; namely, those of Strumpell (?), Westphal, Minkowsky, Nonne, and Eberle; the clinical condition described by Erb was based in a combined system-disease of spinal cord (without meningeal or noteworthy vascular lesion).

And the conclusion is that the pure form of syphilitic spinal paralysis (of Erb) *may* be, and sometimes *is*, conditionated solely by a *primary combined system-disease of the cord.*

The symptomatology of this clinical form was so fully stated in my previous paper[1] that nothing need be added here.

When the clinical picture of Erb's syphilitic spinal paralysis is dependent upon a transverse spinal lesion it is essential that this last be *incomplete*. As Bastian showed, with *complete* transverse destructive lesion of upper part of cord the muscles of lower part of frame are flaccid, and the deep reflexes abolished. Spiller summarises Bischoff's attempted explanation of this fact:—"*In rapidly developing*, complete, transverse lesion of the cervical and upper thoracic cord the vaso-motor nerves are also paralysed. As a result of this, the abdominal blood-vessels are distended, and there is anæmia of the lumbar cord, with loss of tendon reflexes, which becomes a permanent loss if organic changes develop. In cases of *slow development* the cause of the absence of the reflexes must be sought in such conditions as secondary degeneration of the muscles and nerves, or in involvement of the lumbar cord by secondary degeneration." (No italics in original.)

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[1] BRAIN, Spring Number, 1895, p. 147 A to 147 O.

Syphilis of Spinal Cord producing more or less completely the symptoms of Brown-Séquard's paralysis, and viewed from the clinical standpoint.

In the *Third Part* of a former communication I[1] wrote under the section-heading of "*Syphiloma of Spinal Meninges and of Spinal Cord*" that when there is one gumma of fair size, or two or more such, "the solitary gumma is apt to affect, more or less, one side of the cord, especially the posterior column and the lateral, and thus may give rise more or less completely to the symptoms of Brown-Séquard's paralysis, namely, that due to a transverse lesion destroying at some level one lateral half of a transverse section of the cord. Gummatous meningitis, or a gumma even when distinctly meningeal in origin, may, by pressure, produce the same effect as if seated in the cord. The Brown-Séquard type of spinal paralysis (namely, paralysis and hyperæsthesia on the same side, below; anæsthesia on the opposite side, below) may also follow a focus of softening affecting one lateral half of the area of a transverse section of the cord, and due to the blocking of a syphilitic vessel, or of several. Moreover, that type of paralysis is the extreme example of that of which we often see minor degrees in syphilis of the spinal cord; namely, for example, a much greater degree of paraplegia in one leg than in the other, of sensory symptoms on one side than on the other."

Examples were referred to. And in the next section, on "*Syphilitic Disease of Larger Spinal Arteries and Veins,*" the subject was continued in the following terms:—"As just mentioned, the syphilitic disease of blood-vessels supplying the cord may lead to obstruction of the vessels and local softening, with symptoms corresponding to the exact site of this focal alteration; as, *e.g.*, when, affecting one side of the cord, it produces, more or less fully, a palsy of the type made by experimental transverse lateral hemisection of the cord. Subsequently, the secondary degenerations, descending and ascending, consecutive to the local lesion, will modify this, and any other clinical aspect, by superaddition of the clinical results they occasion." In both passages, these explanations of the symptoms followed the conclusions drawn from his experiments by Brown-Séquard.

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[1] BRAIN, Spring Number, 1895, p. 143, *et seq.*

Recently Strauss[1] has reported (from Senator's clinic) a case of Brown-Séquard's paralysis, on a syphilitic basis. On the left side, below the lesion, were motor paralysis and slight hyperalgesia. On the other side, total anæsthesia; analgesia; and loss of thermic sensibility. The feeling of contact was perverted as well as lessened. Both cutaneous and deep tissues were analgesic. At the upper limit of the region of insensibility to impressions of pain and temperature, on the right side, was a hand-wide hyperæsthesic half-girdle or semi-zone.

In the Society discussion on the case, the occurrence of Brown-Séquard's paralysis from hæmato-myelia was emphasised; and, so far as it went, was quite in accordance with part of the view expressed in my article several years previously, partly in the sentences quoted a moment ago, and partly in those following them (*loc. cit.*, p. 144).

At all events, whatever the accompanying lesions and their precise position may be, when finally decided by experiment and observation, the *clinical* symptom-group which we term "Brown-Séquard's paralysis," or some modification of it, or clinical syndrôme analogous to it, is not infrequent in nervous syphilis. And it is upon the *clinical* symptom-group that emphasis is placed here, and not upon the exact anatomical site, or pathological character, of the lesion which gives occasion for the Brown-Séquard syndrôme, or its modifications, or analogues.

X.—THE RELATIONS BETWEEN SYPHILIS AND TABES.[2]

Lately, Virchow is reported to have kept an open mind on the relations of tabes and syphilis; the production of tabes dorsalis by syphilis not being proved, inasmuch as it holds as a general rule that nothing in the evolution of tabes dorsalis is comparable to what is known as visceral syphilis, and no symptoms of visceral syphilis are usually observed in tabetics, nor tabetic lesions in grave cases of visceral syphilis. Yet there being strong evidence in favour of a connection between syphilis and tabes.

Drennen questions whether syphilis is an active cause or underlying condition in the production of tabes dorsalis; points to other causitive factors; finds syphilis rife, tabes very rare, in Japanese and Negroes; and surmises that over-use of K.I. may give rise to tabes.

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[1] *Berliner Klinische Wochenschrift*, Jan. 19, 1898.

[2] See also fuller consideration, BRAIN, Summer Number, 1895, p. 354.

Of 108 tabetics in Leyden's practice, Storbeck made out only about 20 per cent. to be certainly syphilitic—a proportion even less than that found by Westenhoeffer, he states. According to Naegeli, in the same community the actual occurrence of syphilis in the general population was 22½ *per cent.;* or about the same as in the tabetic element of it.

The sexual erethism of early tabes readily exposes to luetic infection; an incidental effect may thus be mistaken as the productive factor. Moreover, it is urged that true tabes is not cured by K.I. and Hg., the recoveries mistaken as such being cures of true cord-syphilis simulating tabes dorsalis; and that cases have occurred of true tabes, subsequent syphilitic infection, and the adoption of anti-syphilitic treatment with benefit to the latter disease but not to the antecedent tabes. Lewin is cited as finding no case of tabes in 800 female syphilitics; Gluck, also, as being against the theory of tabes dorsalis from syphilis; and as being so because of the occurrence of luetic infection in tabetics; because of the rarity of tabes in Negroes and Asiatics, in whom syphilis is extremely common; and because of the absence of any case of tabes in 3,000 syphilitic Bosnians.

On the other hand, in a previous section, we have mentioned the claim that in tabes dorsalis, syphilitic myelitis, and general paralysis there is a preponderance in certain locations of a chronic inflammatory process (meningo-myelitis) present in all three, and that although we are not in a position to assert that the pathological changes are *always* from one and the same cause, yet the vast majority are directly connected with syphilis. But tabes dorsalis does not seem to fall into line here, nor the claim to be fully valid.

As stated in my previous communication, there are "cases linking tabes with spinal cord disease clearly due to the specific lesions of syphilis. Such, especially, are the cases of syphilitic lepto-meningitis invading the cord, particularly its posterior columns and horns, sometimes producing a more or less tabetic, or quasi-tabetic, posterior sclerosis, and diagnosed during life as examples of tabes, or of anomalous, atypical, or pseudo-, tabes. Of such kind are cases (in Part III.) cited from Ewald, Eisenlohr, Kuh, Dinkler, Brasch, Schulz, Siemerling, and one or two of the present writer's cases.

"Moreover, syphilitic spinal lesions have been found associated with the recognised lesions of typical or classical tabes. In this communication (see Parts I. and III.) have already been described in abstract two such cases, one from Raymond and one from Hoffmann, and the second of the present writer's three cases, mentioned in this Part IV., was probably of that nature."

Somewhat similar were cases by Sachs and Nageotte.

A case of cerebro-spinal syphilis by Cassirer, and summarised in a preceding section, is also relevant here. In it, *inter alia*, was spinal syphilis, vascular and invading the cord substance, *plus* a posterior root-zone degeneration, of tabetic type, in the lower part of cervical cord on the left side. As well, also, was there degeneration of the "intra-medullary" part of the right fifth, and of the left fourth, cranial nerves. As regards posterior root-zone, Nageotte's case was similar. Cassirer's case was not one of tabes dorsalis proper; the tabetiform lesions were unilateral only; and of them that of the cord was cervical, and not chiefly lumbar as in regular tabes dorsalis. Nor was Lissauer's tract affected. Yet the case seems to support a connection between syphilis and at least some cases of tabes.

In several cases reported, with tabetic change *plus* syphilitic pathological lesion, the tabetic is in first line, but genuine syphilitic lesion is found, also. In the case just cited, the characteristic genuine syphilitic appearances dominate, and besides them we find an appearance the same as part of that typical of tabes dorsalis in the early stage: viz., a degeneration of posterior root-zone.

In a former article[1] on this subject I stated that whereas in tabes the grey degeneration is somewhat analogous to, or partly identical with, secondary Wallerian degeneration, yet "probably it is more precisely akin to peripheral neural degeneration or neuritis, from the toxines of infectious diseases, or from chemical poisons—in a general word, from toxæmic states, including infections, dietetic and chemical poisons, certain ptomaines, and leucomaines; but herein it shows close kinship with general paralysis.

"Therefore I think that tabes may be accepted as at least mainly of toxæmic origin, whether from:

"Auto-intoxications; altered metabolism; lithæmia, gouty and rheumatic blood-states generally, diabetes, anæmia perniciosa; or from:—

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[1] BRAIN, Summer Number, 1895, p. 371.

"Chronic infections; as syphilis, tuberculosis, leprosy; or from:—

"Acute infections, possibly; (such as diphtheria, typhus, enteric fever, influenza, variola); or from:—

"Ergot — lead — mercury? — iodine? — carbonic oxide?" (Slightly rearranged here.)

Indeed, what is stated now on tabes may be taken as an addendum to the whole of PART IV. of the former communication, p. 354 to 373; which contains many points, repetition of which it is desirable to avoid.

Among the diseases just mentioned is *diabetes*, in which degenerative cord-changes are sometimes observed. As mentioned in my abstract,[1] these when present usually affect parts of postero-median and postero-external columns. Also may peripheral neuritis or nerve degeneration affect the diabetic. And either owing to the changes affecting the cord, or to those implicating peripheral nerves, as the case may be, the knee-jerks may become lost.

A case of acute yellow atrophy of liver in the secondary stage of syphilis, with pathological changes in the spinal cord, was published by A. Goldscheider. The cord presented both diffuse and localised changes, the latter chiefly affecting part of posterior columns and of back of lateral columns. The morbid process preferably affected middle root-zone of posterior columns. The focal changes were at arterial terminations in the cord-substance; and there was hypertrophy but not hyperplasia of the glia elements, the sustentacular tissue being swollen but without nuclear increase. The vascular alterations were of prime importance. The presumption was that the spinal changes were due to the toxic state which produced the acute yellow atrophy of the liver, and, with some differences, on the whole resembled the spinal cord-alterations liable to be found in fatal anæmias.

XI.—INSULAR SCLEROSIS AND SYPHILIS.

In the chronic infections (of which syphilis is one) the nervous system is apt to be affected by the production of forms of arteritis, and patches of sclerosis of scattered, disseminated distribution. This we have already mentioned more than once. It is surprising, therefore, that insular sclerosis has usually, although not universally, been deemed to be entirely without syphilitic connection.

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[1] BRAIN, Spring Number, 1895, p. 179.

The irregular incidence of syphilitic lesions, their nature, the easy possibility and known occurrence of sclerosing relics in their track; all make syphilis a very appropriate agency to suspect of setting insular sclerosis in train. And in many cases at least insular sclerosis is the end, the final page, of a long pathological chapter.

The dominant belief on the ætiology of insular sclerosis makes the most important factor in its production to be infectious disease: *e.g.*, enteric fever, chronic malaria, variola, measles, scarlet fever, pneumonia, erysipelas, diphtheria, whooping-cough, dysentery, influenza.

The pathological change does not appear to be usually a primary overgrowth of interstitial tissue. Nor is a primary wasting of nervous parenchyma—to which the interstitial change is secondary—very frequently described, or assumed, in insular sclerosis. On the other hand, the evidence of vascular origin of the patches is strong; whether by way of thrombosis, or of multiple embolism, or of an extension of a morbid process in the vessel-walls to the surrounding tissues. The patches of what, finally, is known as an insular sclerosis may be of encephalitis or myelitis—whether originating in inflammatory affections of blood-vessels or not. Nevertheless, Redlich, for one, makes the vascular change secondary to the neuroglial; and Weigert and Strümpell give chief place to the latter.

In several of the facts above-mentioned we seem to find much in common with the known behaviour of syphilis. And in some diseases in which syphilis appears to be a potent factor—*e.g.*, general paralysis—there often are symptoms reminding one of insular sclerosis; in treating of such cases I long ago wrote that "in truth there is a secondary scattered sclerosis in some cases of general paralysis." Patches and small areas of indurative cortical cerebritis—like islands in a sea of the ordinary diffuse cortical change—I have sometimes observed, also.

Von Bechterew has published a syphilitic case resembling disseminated sclerosis, on which Spiller[1] remarks. Very recently, B. Sachs[2] has urged the close likeness sometimes seen between the symptoms of multiple sclerosis and multiple cerebro-spinal syphilis. He reports three cases in exemplification of the difficulties sometimes experienced in distinguishing between the symptoms of multiple sclerosis and those of multiple cerebro-spinal syphilis; these difficulties being especially experienced in cases beginning with spinal symptoms. As distinguishing points of detail, he observes that in multiple sclerosis paræsthesiæ are more frequent, and pain in syphilitic affections: nystagmus a characteristic in disseminated sclerosis, but rare in brain syphilis: complete oculo-motor nerve paralysis frequent in cerebral syphilis and often precedent to other of its symptoms, but very rare in multiple sclerosis, and still more rarely precedent to other symptoms by a year or more: and that complete immobility of the pupils in exposure to light and during accommodation is characteristic of a general syphilitic affection. This last doctrine Mr. Jonathan Hutchinson has been teaching for a generation; and it is interesting to see this long-established conclusion coming back to us from the other side of the Atlantic as something new. The greater frequency of optic neuritis in syphilitic affections is also mentioned in contrast with the primary (? W. J. M.) optic-nerve atrophy so frequent in multiple sclerosis; and so is the far greater frequency and more complete degree of remission in ordinary cerebral syphilis than in multiple sclerosis. Yet it is possible that the sclerotic patches may follow syphilitic inflammatory patches. The difficulties of the subject are increased by another fact to which he draws attention, that in "one case the disease had for a long time the characteristics of multiple sclerosis and now is a typical instance of paralysis agitans; in the other the symptoms of paralysis agitans were the first to appear and still exist, but those of multiple sclerosis have been superadded."

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[1] *New York Medical Journal*, September 25, 1897.

[2] *Philadelphia Medical Journal*, February 5, 1898. (See also Digest on Sclerosis, in BRAIN, 1898, p. 267.)

And I conclude that if syphilis is not capable of producing insular sclerosis, it ought to be.

Reviews.

Die Geschwülste des Nervensystems; Hirngeschwülste, Rückenmarksgeschwülste, Geschwülste der peripheren Nerven—Eine Klinische Studie. Von Dr. Ludwig Bruns. Mit 31 Abbildungen im text. Karger, Berlin, 1897 (Williams and Norgate, London), 12s. Pp. 388.

Although from the striking character of the symptoms cases of tumour of the brain or spinal cord must always have excited great interest, yet in former times this interest lacked practical bearing, partly from the hopeless nature of the illness, and partly from the want of sufficient data on which to make an accurate diagnosis of the tumour during life. Three causes have within late years greatly stimulated investigation in this field; first, the use of the ophthalmoscope; secondly, the discovery of the localisation of function in certain parts of the brain; the one giving in the presence of optic neuritis a certainty to the general symptoms of brain tumours which could not be obtained before, and the other establishing the reliability of the local symptoms; and thirdly, the demonstration that tumours of the brain and cord could be successfully removed by operation. The rapid advance in the knowledge of these tumours that has followed has resulted from the increased attention thus directed on the subject. Of this knowledge and its most recent developments, this book gives a very complete account, and the reader may turn to it with confidence that nothing of importance has been omitted. The author has taken pains to bring his information fully up to date, and his own experience and wide reading qualify him to deal authoritatively with his subject.

With regard to the pathology of brain tumours the gliomata are attributed to overgrowth of neuroglial tissue, and the author thinks that they must be sharply differentiated from the sarcomata, both on histological grounds and on those of their origin from an ectodermal structure. An important practical distinction from most sarcomata consists in the infiltrating character of the gliomata, so that they may take the form of that part of the brain in which they lie, and from lack of demarcation be incapable of removal. Nerve-fibres and cells within the limits of the tumour may retain their functional capacity, so that the symptoms may be slighter than would seem likely from the extent of the tumour.

Dr. Bruns thinks that traumatism is doubtful as the only exciting cause of a brain tumour, but considers that in syphilis and tubercle it may, by forming a *locus minoris resistentiæ*, determine the seat of a syphilitic or tubercular growth. A traumatism may, however, first call attention to the existence of a tumour, either by causing hæmorrhage into a glioma, or by so altering the surroundings or conditions of any tumour that it takes on active growth, and in each case thus first gives occasion for distinct symptoms to arise. Whilst admitting the cogency of these arguments, however, it seems doubtful whether the frequent history of injury to the head shortly before the appearance of symptoms of a brain-tumour is thus fully explained by them.

A chapter follows on the general effects of the presence of a tumour on the brain itself, on the cranial nerves, and on the ventricles, which will be found of much interest and value.

Dr. Bruns adopts the mechanical theory of the origin of optic neuritis, which he thinks amply sufficient to account for this symptom, supported as it is by recent observations on the subsidence of optic neuritis after operations to relieve increase of intra-cranial pressure. He agrees with Oppenheim's statement that about 90 per cent. of all cases of optic neuritis are due to the presence of an intra-cranial tumour. He accepts the division of the symptoms of a brain tumour into general and focal as one of great practical importance and founded on a sound pathological basis, but points out that the line between them must not be too sharply drawn, as "general" symptoms may under special circumstances have a focal or local significance, *e.g.*, those due to the early appearance of internal hydrocephalus in tumours of the posterior fossa, and on the other hand symptoms usually focal in import may be in some cases "general," as when pressure on the olfactory nerves or optic chiasma arises from increase of the general brain pressure. The characteristic mental change in intra-cranial tumours is considered to be mental dulness or stupidity; marked psychical symptoms are specially frequent in multiple tumours.

Other things being equal, the smaller the growth and the slighter the general symptoms the more certain is the local diagnosis. In the localisation of tumours considerable importance is to be attached to increased tenderness of the skull to percussion. When the tenderness is general over the skull there is likely to be a general increase of internal pressure; this is apt to occur early in the illness in large tumours in any situation, and also in tumours of the cerebellum. Local tenderness, if it exists over the position that other signs indicate as the seat of the tumour, is a guide to its localisation, and tends to show that it lies in or near the cerebral cortex, or in the membranes, though this sign may be present in deep-seated tumours. A tympanitic percussion note, and *bruit de pôt fêlé* are also sometimes met with, and, if these signs are localised, and correspond to the position assigned to the tumour on other evidence, are valuable in diagnosis. The author states that he holds tenderness on percussion, tympanitic note, or *bruit de pôt fêlè*, to be such valuable signs that, if, for instance, other symptoms indicated the motor convolutions as the seat of a tumour, the presence of these local signs over a neighbouring area, say, in the frontal or parietal region, would be sufficient to indicate one of the latter regions as the position in which to trephine. Both signs are most evident and pronounced in children, and are sometimes met with in other conditions than tumour. In the section devoted to the consideration of the symptoms and signs evoked by tumours in special regions of the brain, full details are given of those proper to a growth in each region. It is stated that a tumour of the optic thalamus can be diagnosed with some certainty if symptoms of hemiplegia with distinct disturbances of sensibility, perhaps with hemiopia, and especially with one-sided athetosis and paralysis on the same side of the facial nerve muscles for mimetic or emotional expressions, but not for voluntary movements, are present. Dr. Bruns thinks that it will only very seldom be possible to make a diagnosis of a tumour of the corpus callosum; he denies that there is any especial tendency to mental changes in tumours of the præfrontal area, and when such symptoms are present he attributes them to the fact that a tumour in this region can grow to a large size before it endangers life, an argument which does not apply to those observed cases in which a frontal tumour of moderate size has produced early pronounced mental symptoms. He lays stress, however, as an aid to diagnosis, on the hilarious or humorous mental state, and also on the ataxia which may be observed in cases of tumour in this situation. A useful table which is given of the accompanying symptoms in cerebellar and frontal ataxia shows how closely the group of symptoms may resemble one another in each case.

Whilst he admits the occasional presence of sensory disturbances in tumours of the central convolutions, especially the occurrence of ataxy and disturbance of the power of localisation of the sense of position, he thinks that there is great individual variation in this respect in different persons, the difference being unaccountable unless it is due to the greater extent of sensory (than motor) representation in the cortex, and hence the greater possibility of compensation.

With regard to the diagnosis of the side on which a cerebral tumour is situated, it is pointed out that large tumours may excite symptoms by compression of the opposite hemisphere.

Two forms of ataxy in cerebellar tumours are recognised: (1) the staggering or drunkard's gait, and (2) a gait and movements like those of locomotor ataxy; and ataxy is considered to be more constant in tumours of the vermis than in those of the hemispheres, but cannot be taken as a guide to the position of the tumour in the cerebellum. Tumours of the middle peduncle or of the lateral lobe compressing the middle peduncle are stated to cause movements of rotation of the body round its longitudinal axis.

Cerebellar tumours are noted to be extremely seldom latent.

There is a very good section on the Diagnosis of Tumours at the Base. The author considers that an intra-medullary tumour of the pons can only be certainly distinguished from one lying outside it if the symptom of loss of conjugate movement of the eyes to one or other side has been present from the beginning. With regard to the attainment of an accurate local diagnosis, this can be best accomplished in tumours of the motor centres, of the base (posterior and middle fossa), and of the pons, though in many cases of tumour of the posterior fossa one has to be content with that diagnosis without more accurate localisation. In tumours of the speech regions, cerebellum, medulla oblongata, 4th ventricle, white matter of the occipital lobe, the local diagnosis is less certain. Those of the frontal lobe come in this group, if the local evidence and signs of affection of neighbouring parts be clear. Such a diagnosis is uncertain or impossible in growths of the parietal and right temporal-sphenoidal lobes, of the corpus callosum, centrum ovale, and great ganglia.

In cases in which the general and local symptoms are pronounced, the diagnosis is a certain one in 80 per cent.

With regard to surgical treatment, practically the only treatment in intra-cranial tumour, the author states that taking everything into consideration, 60 per cent. of tumours in which a local diagnosis is possible are unsuited for operation, because they are not accessible to the surgeon. In about 32 per cent. of cases of intra-cranial tumour a certain general and local diagnosis is possible, and the tumour is seated in an accessible position. The *exact* conditions present can, however, never be ascertained until the tumour is exposed, and unavoidable hindrances due, for instance, to the size and extent of the growth, its situation and character, must be considered. The 32 per cent. is thus reduced to about 8 per cent. of all cases; in this 8 per cent. the position of the tumour is correctly diagnosed, it is accessible, and there are no hindrances at the operation to complete extirpation. Deducting again from this cases in which unavoidable surgical accidents occur, and the 4 per cent. of fully successful cases given by Oppenheim is nearly reached. These statistics were of course compiled before the recent results obtained at the National Hospital were published by Professor Ferrier; these give a much more favourable aspect to operation.

Taking all things into consideration, Dr. Bruns dissuades from operation in cerebellar tumours. He speaks strongly in favour of trephining as a palliative measure in cases of irremovable intra-cranial tumour, especially if blindness threatens.

The whole question of operative treatment, and the advice to be given to patients in this respect, is treated in a masterly manner. Dr. Bruns would put as clearly as possible the exact conditions before the patient and his friends, and in a suitable case would advise an operation, but would not urge it. With regard to intra-cranial syphilis he says that in his experience the lasting cure of gummatous processes by anti-syphilitic remedies is much more seldom than is generally stated in the text-books; in most cases the improvement is temporary.

The section of the book devoted to Tumours of the Cord is also a very complete one, and gives a full account of their symptomatology and pathology. The regional or segmental diagnosis is entered into very thoroughly, and is brought well up to date; special stress is laid on the importance of Sherrington's discovery of the overlapping in the distribution of the nerve roots; the author thinks that even five roots may in man supply fibres to a single area of sensory distribution. The researches of Head or Thorburn on segmental distribution are also fully utilised. The differential diagnosis between tumours of the spinal column, those within the spinal canal but extradural, and those growing in the cord itself, receives a full discussion. The directions for localising the position of the growth are very clear and good.

Dr. Bruns especially mentions the frequency of cancer of the vertebræ secondary to mammary cancer; these secondary deposits may occur after so long an interval as eight years after a complete and apparently successful operation on the breast. He thinks that the outlook is so hopeless in cases of tumour of the spinal cord that operation is not only justifiable but obligatory in every case in which a tumour of the spinal cord can be diagnosed with certainty. He gives a short account of the published cases of this operation; the first successful one was the memorable case of Gowers and Horsley. In all there are twenty reported cases of operation for an "occult" spinal tumour; it is interesting to note that the diagnosis was correct in every case, and in eighteen the tumour was found at the operation. In six, or 30 per cent., cure or great improvement resulted, in two there was slight or temporary improvement. In twelve cases the patient died, in nine soon after the operation from shock, hæmorrhage or sepsis. Considering the grave nature of the disease and the difficulties involved in the operation, this may be considered as a good result.

The concluding portion of the book deals with tumours of the peripheral nerves in the same thorough manner. The classification of neuromas into true and false neuromas is shown to be misleading, in that these growths spring from the epi-, endo-, or peri-neurium, most often from the latter, and are all, therefore, false neuromas in Virchow's sense. It is not denied that a growth of nerve substance sometimes takes place in these tumours, but with the exception of many cases of amputation neuroma, it almost always takes a small part in the construction of them. The nerve fibres are most apt to be damaged in sarcomas; in ordinary neuromas, although the nerve bundles are separated from each other and broken up, the nerve fibres often retain their medullary sheaths, and presumably their functional capacity for a long time. The important fact is mentioned that malignant neuromas (sarcoma) often start on small peripheral twigs, and the recurrent growths take place nearer and nearer to the centre until they reach parts no longer accessible to the knife.

I have only alluded to a few points of present interest, but the book itself will be found to give a very thorough account of the whole subject in all its bearings, and will well repay a careful perusal. It is written in a clear style, in a highly interesting manner, and has evidently been well thought out. The author gives full credit to the work of others, and his cordial appreciation of the labours of English-speaking observers, which he repeatedly quotes and refers to, must be particularly gratifying to us.

The absence of an index is a serious defect to the book as a work of reference, for which it is otherwise very well qualified to serve. This will no doubt be rectified in a future edition.

J. Michell Clarke.

Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie. I. Jahrgang. Bericht über das Jahr 1897. 1 vol. large 8vo, pp. 1508. Karger, Berlin, 1898.

This work has just reached us, whilst the last sheet of this number of Brain was going through the press. It will prove an indispensable companion to every working neurologist, whose labour in looking over the literature of the subject in hand will be greatly facilitated. It is intended to give a full *résumé* of all the neurological contributions of any value published during the past year; and the names of Drs. Mendel, Flatau and Jacobson, which figure on the title page, are a guarantee that this aim will be reached. The list of working contributors is also promising. The great advantage of a yearly *Bericht* over a *Centralblatt* is that the papers abstracted are arranged under distinct headings, and that the work being done more systematically, the reading of every chapter gives a much clearer notion of the progress made during the year. On the other hand the information is necessarily somewhat belated, so that it is by combining the two sources of information that the student will obtain all the profit derivable from these most praiseworthy efforts of our laborious German collaborators in the field of neurology. It would obviously be a hopeless task to try, in a short notice, to give an idea of the enormous mass of material accumulated in these 1,500 pages. The reader will, however, have no difficulty in finding what he wants, for the divisions and sub-divisions of the work are clear, and the type and paper fairly good. The subjects embrace the whole field of neurology and psychiatry, from the methods of colouring of the nervous elements to questions of criminality and the like. It only remains for us to thank most heartily the editors, authors and publisher who have given us a work of such supreme practical value in our labours, and to hope that their efforts will be further encouraged by the reception this *Jahresbericht* will receive at the hands of Neurologists.

A. de W.

BRAIN.

PART IV., 1898.

Original Articles and Clinical Cases.

PATHOLOGY OF A CASE OF FRIEDREICH'S DISEASE.

With a Summary of previously reported cases.

BY H. MACKAY, M.D.

THE case of Friedreich's disease, the morbid anatomy of which I here report, was one of a family described clinically in the *American Journal of the Medical Sciences*, vol. cviii., 1894, p. 151.

To save the trouble of reference, I append here a brief note of the clinical condition of the patient during life.

H. W., male, unmarried, aged 26 at the time of examination, was one of a family, three members of which, *i.e.*, two sisters and himself, have been under my observation for Friedreich's disease. A fourth member, a sister, had already died from the same complaint. There is no hereditary transmission of the disease, but various spinal neuroses are present in collateral relatives of the patient, and a second cousin probably died of the same disease.

The patient suffered from measles and scarlet fever in his sixth year, soon after which weakness of the legs and staggering appeared. At twelve he was unable to move about; at fourteen the arms began to be attacked, and by his twenty-second year these limbs were helpless.

He came under my notice about this time and presented a typical picture of the later stage of the disease. His power of voluntary movement was limited to slight motion at the joints of the upper limbs, raising and rotation of the head, and some control over the muscles of expression. He was also able to roll over in bed, and to raise the trunk on the elbows when supine. His deep and superficial reflexes were abolished, with the exception of normal pupillary reactions to light and accommodation. His sensation was everywhere well preserved, and his special senses acute.

Coördination had been much impaired before the limbs became fixed. He had lateral nystagmus and frequent twitchings in the facial muscles. His speech was laboured and explosive. His intelligence was good, and he had complete control over bladder and bowel. The spine showed a lateral curvature. The optic discs were normal.

The patient suffered from asthma, and from cardiac enfeeblement, but without recognisable dilatation or valvular disease.

In the winter of 1897, when 30 years of age, he contracted measles for the second time. Symptoms of collapse speedily supervened, with cyanosis and heart failure from which he died.

The autopsy, some twenty-eight hours after death, showed the following. Permission to open the chest was not obtained.

Cranium.—Much adhesion of Pacchionian bodies, otherwise no abnormality.

Membranes.—Normal; no excess of fluid in subdural space.

Brain.—To naked-eye inspection normal. The grey and white matter were natural on section, and the convolutions well marked.

Cerebellum.—Well developed and of average size. Measurements in the three chief planes:—11·8 c.m. × 5·3 c.m. × (between vermiform processes) 4 c.m. Schultze gives, as average figures, 11·5 c.m. to 12·5 c.m. × 5·25 c.m. to 7·5 c.m. × 3 c.m. to 4 c.m.

Medulla.—Visibly smaller than normal. Vagi, hypoglossi, and other cranial nerves appear to be normal.

Cord.—The dural sac is loose and baggy, and the contained fluid excessive in amount. The latter looks like normal cerebrospinal fluid. The dura appears thickened, but the pia has no abnormal appearance. The cord is evidently shrunken in size. On section the posterior column is everywhere greyish and translucent, and the same appearance is visible in the posterior part of the lateral columns. The posterior spinal roots are visibly shrunken in size. The anterior appear normal.

The contents of the cranium, and the spinal cord, were placed in Müller's fluid, together with portions of the following peripheral nerves: the sciatic and the anterior crural of the right side, the anterior tibial and internal plantar of the left side. From the upper limbs were taken portions of the right median and ulnar, and of the left radial and posterior interosseous. These latter were hardened in Erlitzky's fluid followed by methylated spirit.

For defining the areas of degeneration in the cord and medulla, preference was given to Schäfer's modification of the Pal-Weigert method. By this process, myelin-bearing fibres, whether healthy or degenerating, stand out in black or blue-black against the white ground of sclerosed or non-medullated tissue. For differentiating healthy from degenerate myelin, Hamilton's modification of Marchi's process was used. For investigating the neuroglia, to which, since the appearance of Déjérine and Letulle's observations[8] considerable interest has attached, attempts were made to use Weigert's—now classical —1895 method,[33] but owing to the hardening of the material in Müller's fluid, the attempt was not successful. Mallory's 1895 method[34] failed also for the same reason. Better results were obtained by a preliminary stain in Van Gieson's fluid, followed by Mallory's phospho-molybdic or phospho-tungstic hæmatoxylin.[35] By this means an excellent triple stain is obtained, in which the dark axis-cylinders and glia cells are well distinguished from medullary sheaths (yellow) and connective tissue other than glial (red-violet). For the peripheral nerves Schäfer's method answered admirably, while for axis-cylinders aniline blue-black, benzopurpurin, or Mallory's hæmatoxylin was used. For the spinal ganglia toluidin-blue and eosin, hæmatoxylin and eosin, or picro-carmine were employed.

The actual changes found were as follows :—

CORD.

T. S. Lower Lumbar (L. V. and IV.)

Posterior Column.—There is well marked sclerosis, most marked in the posterior part of the column. At the level of the 5th lumbar this involves the posterior third of the column. The anterior third, next to the commissure, shows a large number of well-preserved fibres, but also a few swollen and degenerate ones. The middle third of the column shows a gradual transition between the conditions described. In the posterior third very few stained fibres can be seen, and these are scattered at wide intervals throughout the sclerosed area. A band of comparatively well-preserved fibres lies along the mesial side of the posterior horn on each side, separating the sclerosed portion from the grey matter. This, which is continuous with the well-preserved fibres lying dorsal to the commissure, evidently represents Marie's cornu-commissural tract.

Of the posterior root fibres, those entering the gelatinous substance (middle group of Lenhossek), those entering Burdach's column (mesial group), and those occupying Lissauer's border-zone are all intensely degenerated, the course of individual fibres passing horizontally being marked generally by isolated droplets of myelin, while those transversely divided display swollen sheaths and an absence of axis-cylinders.

The sclerosed area includes the septo-marginal tract of Bruce and Muir and the central oval tract of Flechsig, neither of which contain healthy fibres.

At the level of L. IV. the dark stained fibres occupy only the anterior fourth of the posterior column. The remaining three-fourths are degenerated, the posterior portion the most severely. About the centre of the posterior column, however, a small, linear tract of fairly well-preserved fibres appears, bordering the posterior fissure. This has not the oval shape of Flechsig's *centrum ovale*, and probably corresponds to a portion of Bruce's septo-marginal tract.

Lateral Column.—At the lower level, L. V., there is apparent under the low power a lightening of the myelin stain in a triangular area occupying the posterior part of the column, but separated from the posterior horn by the well-preserved fibres of the lateral limiting layer. At the higher level, L. IV., the degeneration is more pronounced, and corresponds with the position of the crossed pyramidal tract. In connection with Marie's view[36] that the degenerated area in the lateral column does not represent the crossed pyramidal tract, it was noted that the degeneration affected equally the large and the small fibres of the area. It is equally true, however, that many large fibres remained unaffected throughout the tract. As will be seen afterwards, the size of the lateral sclerosed area diminished from the mid-dorsal region upwards, but it increased from the lumbar to the mid-dorsal region, and at the latter level well-marked degeneration appeared in the fibres of the direct pyramidal tract, which might account for the lateral diminution from that level upwards. There is distinct neuroglial overgrowth present, with an increase in the number of round, granular glial nuclei.

Anterior Column.—Normal.

Grey Matter. Posterior Horn.—There is extensive degeneration both of the vertical (ascending) and of the transverse (entering) fibres in the caput cornu.

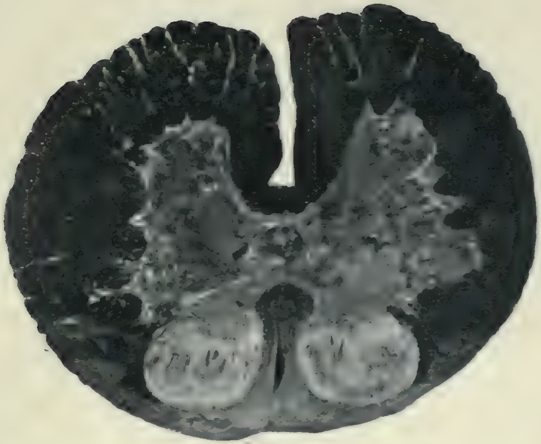

FIG. 1.

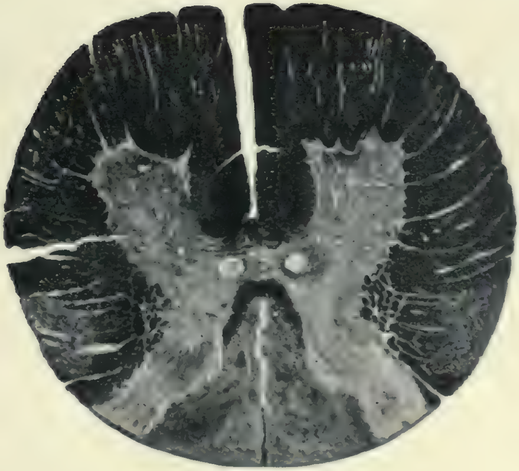

FIG. 2.

There is no trace of any healthy nerve cells, while the adjoining large cells of the intermediate grey substance are well preserved. The reticulum of fine medullate fibres in the substance of the horn is fairly but not well, preserved.

Anterior Horn.—The large motor cells with their processes, and the fibrillar plexus are well preserved.

The Central Canal is occluded by a mass of small, round cells, which take on nuclear stains deeply. No trace of ependymal epithelium remains.

The posterior roots, spinal ganglia, and anterior roots will be described in detail later. It is sufficient here to say that the posterior roots were intensely sclerosed.

Upper Lumbar and Lowest Dorsal L. I. to D. XII.

Posterior Column.—At this level the postero-median fissure is for the first time recognisable. Previously—at lower levels—it had only been obscurely suggested, and had contained no prolongation from the pia. There is as yet no differentiation of Goll's from Burdach's column.

The area of stained medullate tissue in the P.C. is now limited to the tract of cornu-commissural fibres, which fill the apex of the posterior column, and occupy its lateral margins close to the grey matter. This area, however, contains a considerable number of swollen and otherwise degenerate fibres.

Lateral Column.—The degeneration of the C.P. tract is now well marked. It occupies a triangle, whose posterior base, separated from the posterior cornu by the thick layer of the lateral limiting zone, extends inwards from the periphery for about half the length of the posterior cornu. The apex reaches anteriorly to the level of the posterior commissure.

Anterior Column.—Normal.

Posterior Horn.—At the base of the posterior horn two or three shrunken cells can with difficulty be made out. There is a thin plexus of fine myelinised fibres anterior to the substantia spongiosa. The trabeculæ of the latter are thickened and numerous large Deiter's cells are visible. The vertical fibres, as well as the entering root fibres are degenerated.

Anterior Horn.—Apparently normal.

Clarke's Column.—This appears on each side as a faintly indicated white disc. Its posterior half is sclerosed. At its anterior pole, on each side, a thin plexus of fine fibres is visible, together with a few shrunken cells.

Lower Dorsal.—D. X.

Posterior Column.—There is still no trace of the differentiation of Goll's from Burdach's column. The whole breadth of the posterior column is occupied by the greyish white area of sclerosis. Some dark stained medullate fibres still occupy the apex and lateral boundaries of the column—in the site previously occupied by the cornu-commissural tract (ascending fibres from posterior roots ?)—but these show an increasing number of degenerating fibres.

Lateral Column.—The patch of lateral sclerosis now takes on an extension forwards along the periphery of the column, the increase probably corresponding to the area of the direct cerebellar tract. This marginal degeneration reaches forward to a level corresponding to one-third of the peripheral circuit from posterior root to anterior fissure. The anterior portion of the degenerate tract is much less affected than the posterior.

Anterior Column.—There is now some disappearance of nerve tubes, with swelling and disintegration of others, and increase of interstitial tissue, in the anterior tips of Turck's columns on each side, where these border on the anterior fissure.

Posterior Horn.—The condition is the same as that described in the lumbar region.

Anterior Horn.—There is apparently some diminution in the number and volume of the internal group of motor cells.

Clarke's Column is completely sclerosed, both as to cells and fibres.

Mid-Dorsal. D. VII. to D. IV.

Posterior Column.—The narrow strip of medullated tissue bounding the commissure and posterior cornua shows a progressive loss of healthy fibres.

Lateral Column.—The degeneration in this column reaches its maximum extent about D. V. and at that level occupies a triangle, elongated anteriorly—whose base covers rather more than half the width of the lateral column, while the apex reaches the level of the summits of the anterior cornua. The lateral limiting layer is still preserved.

Anterior Column.—The sclerosis of the tips of Turck's column is well marked.

Posterior Horn.—This seems to be shrunken in size—the caput cornu reaching less than half way to the periphery. Its condition is as already described.

Anterior Horn.—No fresh changes are visible.

Clarke's Column.—Sclerosed.

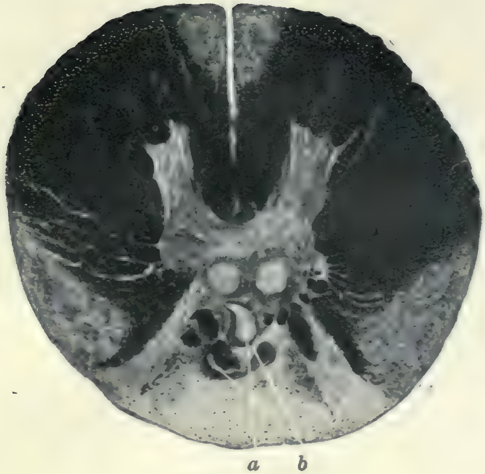

Fig. 3.

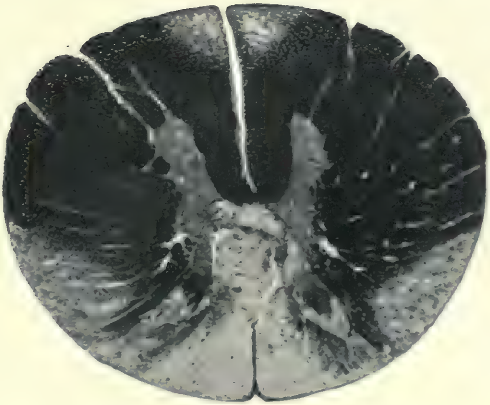

Fig. 4.

From D. VI. to D. III. an abnormality was present in the posterior column. A rounded patch of dense, sclerosed tissue occupied the anterior angle of the column, close to the commissure, displacing the fibres previously present there. These latter were represented in all probability in a ring-like whorl of medullated and partially degenerate fibres, which occupied the middle third of the column, posterior to the sclerosed patch already described. The whorl of fibres enclosed a second circular sclerosed area, and the remainder of the posterior column was occupied by sclerotic tissue as at former levels.

Upper Dorsal. D. II. to D. I.

The sclerosis of the *posterior column* here reaches its maximum, the remnant of stained fibres in the lateral tracts of Burdach's column, and posterior to the commissure, having undergone still further diminution.

Lateral Column.—The anterior marginal extremity of the sclerosed area does not extend so far forward as previously, having reached its maximum extent at the mid-dorsal region.

Anterior Column.—There is well-marked and symmetrical sclerosis of the anterior third of Turck's column. Degenerate tubes and increased neuroglia can be made out in the remainder of the direct pyramidal tracts on each side.

The shape of the cord departs from the normal here, being more oval than is usual at this level. This may possibly be due to antero-posterior shortening, caused by sclerotic changes in the anterior and posterior columns.

At the upper dorsal region, viz., D. I., there is apparent for the first time a distinct differentiation of Goll's from Burdach's column. A further change that takes place from this level onwards is the appearance of an increasing number of healthy, medullated fibres in the postero-external fields of Burdach's columns.

Lower Cervical. C. VIII. and VII.

Posterior Column.—The increase in the healthy fibres in Burdach's column now extends over the whole of that tract, and serves to distinguish it from Goll's tract. The increase in healthy fibres is also apparent in Lissauer's tract, which, though still much degenerated, yet contains more black-stained fibres than previously. Goll's column remains sclerosed up to the posterior commissure, and is shrunken to a narrow streak on each side the posterior fissure.

Lateral Column.—The position and degree of the sclerosis are as before. The anterior point reached by the degeneration in the D. C. T. is nearly on a level with the anterior commissure. In the upper sections of this region, however, there seems to be an increase in the thickness of the neuroglial trabeculæ in the position of Gower's tract, but without any distinct degeneration of the tubes.

Anterior Column.—There is well-marked and symmetrical sclerosis of Turck's column, appearing as a bluntly wedge-shaped tract on each side of the anterior fissure. The rest of the ground fibres are normal.

Posterior Horn.—This is distinctly atrophied and shrunken. There are a few nerve-cells in each cornu, but they appear below the normal size. The gleia cells are numerous and deeply stained. The preserved fibres with well-marked axis-cylinders, however, appear to be more numerous than previously.

Anterior Horn.—The inner or mesial group of cells has undergone diminution in number, and in size of the individual cells. The remaining groups, however, contain well-developed cells and processes.

Middle of Cervical Swelling. C. VI. to V.

An abnormality is again present in the posterior part of the cord at this level.

It commences at the level of C. VII. as a circumscribed oval-shaped area of sclerosed tissue, which occupies a position between the neck of the left posterior cornu and the adjoining portion of Burdach's column. The sclerotic area increases in size with successive sections, and reaches its maximum at the level of C. VI. In the position it occupies it displaces inwards towards the mesial line the streak of medullated fibres which has been described as bordering the internal margin of the posterior cornu. Its outer side is in contact with the lateral reticular formation. Completely sclerosed at its first appearance, at higher levels its area is traversed by scattered medullated fibres, a few of which are healthy but the greater number degenerated. By the level of C. IV. all traces of the abnormality have disappeared.

Posterior Column.—The condition is similar to that described previously. Burdach's column continues to receive an increasing number of healthy fibres, and also some degenerate ones. The healthy fibres are aggregated for the most part in the postero-external fields of the tract, adjacent to the posterior roots, and in Lissauer's tract. Goll's column also contains more myelinised

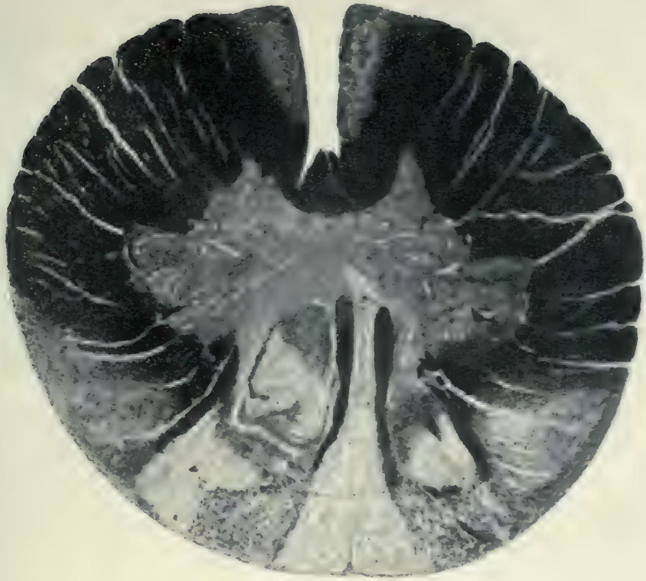

Fig. 5.

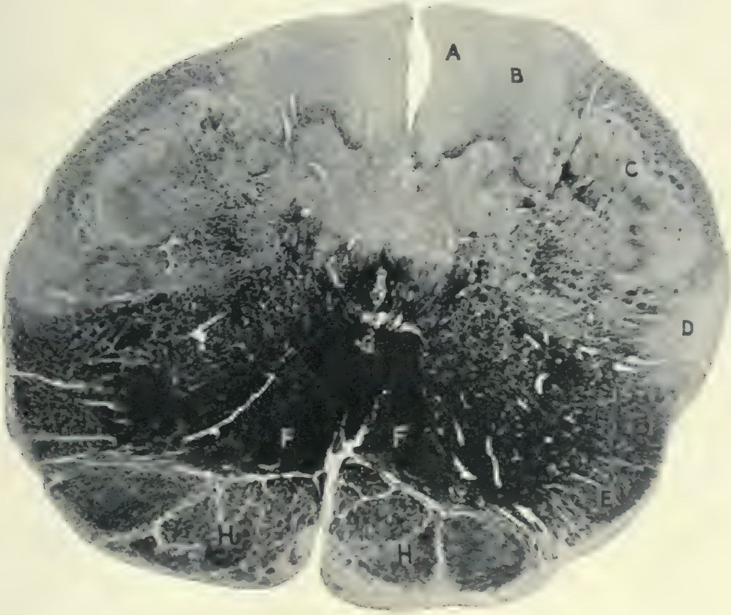

Fig. 6.

fibres than at former levels. These pass obliquely through the column towards the commissure, and many of them are degenerated.

Lateral Column.—The sclerosed area maintains its former position. The anterior extremity reaches further forward on the left side than on the right.

Anterior Column.—As before. The direct pyramidal tract on each side is symmetrically sclerosed. There is probably some increase of the neuroglia in the anterior ground fibres.

Posterior Cornua.—The abnormality on the left side has been already described. On the right side there is some improvement in the fulness of the fibrillar reticulum anterior to the substantia spongiosa.

Anterior Cornua.—There is asymmetry of the anterior horns, the right being larger than the left.

Upper Cervical. C. IV. to III.

Posterior Column. — Goll's tract is sclerosed throughout. Several fibres in advanced degeneration pass through it antero-posteriorly. These are more numerous near the commissure. Burdach's column, in its postero-external triangle, and Lissauer's tract contain a fair number of myelinised tubes, some of which contain healthy axis-cylinders.

Lateral Column.—The degenerate area is smaller in size than previously. The complete degeneration is limited to a small, oval area, which does not now reach to the periphery, and whose anterior limit does not pass beyond the level of the posterior commissure. The fibres of the D. C. tract, however, still contain many degenerated tubes.

Anterior Column.—Turck's columns still sclerosed.

Posterior Cornua.—Still degenerated and shrunken. The abnormality on the left side has now disappeared. Perhaps the fibrillar plexus is more stained than previously.

Anterior Cornua.—The right is still larger than the left. Both contain some shrunken cells in their anterior portions. The lateral groups of cells on each side are large and well developed.

RESUMÉ OF CORD-CHANGES.

Complete Sclerosis of Goll's Column from the sacral to the upper cervical region, most pronounced in the upper dorsal region.

Sclerosis of Burdach's Column (only recognisable as such above the level of the upper dorsal), the degeneration being constant in the portions adjoining Goll's column, and being less marked below in the parts bounding the commissure and the posterior cornua, and above in the postero-external fields.

Sclerosis of Lissauer's Tract less marked in the cervical region.

Sclerosis of the C. P. Tract, from lumbar region upwards, reaching its maximum at mid-dorsal level, and thereafter diminishing.

Sclerosis of D. P. Fibres, from mid-dorsal region upwards, increasing from below upwards.

Sclerosis of D. C. Tract, from lower dorsal region upwards. Most marked below.

Sclerosis of Posterior Horns and Posterior Roots throughout. Anterior horns well preserved, with the exception of a few cells of the mesial group and anterior group. Almost total escape of the anterior roots (to be described later).

Complete Sclerosis of Clarke's Column.

The condition of the nerve roots requires separate mention.

(A.) *Posterior Roots and Ganglia.*

These were examined: (*a*) above the ganglia; (*b*) in the ganglia; (*c*) below the ganglia; (*d*) in the mixed trunks.

The specimens were taken from the lumbar and dorsal region. I regret that the cervical roots were not preserved, all the more because the condition of the intra-medullary root-fibres in that region suggests that they would have shown, perhaps, a state of better preservation than those of the lower level. Cross-sections of the extra-medullary roots, however, were frequently included in sections of the cord in the cervical region, and an examination of these showed no marked difference between these roots in cross section and those belonging to other levels of the cord.

(*a*) The posterior roots between the ganglia and the cord showed extreme degeneration. Both by Marchi's and by Schäfer's method, only a very few dark-stained tubes of normal calibre were present in each root. In the unstained spaces between these larger fibres were seen fine small nerve-fibres, empty sheaths and connective tissue. The fine nerve fibres, probably the "embryonal" fibres of Auscher and Déjérine,[10] were frequently closely packed, in bundles of from six to twenty fibres, the individual

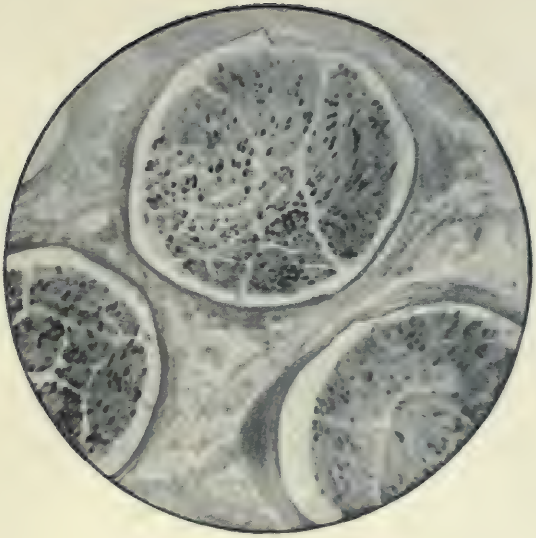

Fig. 7.

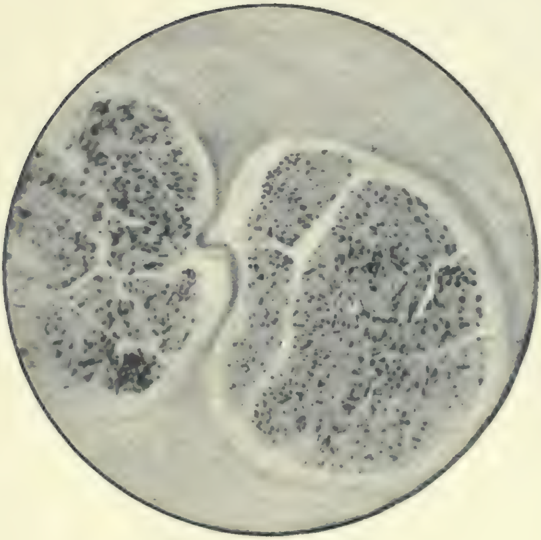

Fig. 8.

tubes measuring from 3 to 8 in diameter. Extremely fine, but well-stained axis-cylinders were present in most of them, and generally a very thin myelin sheath, but the latter remained unaffected by myelin stains such as Schäfer's and Pal-Weigert's. In others, the myelin seemed disintegrated and granular, and the axis-cylinders were sometimes hypertrophied, sometimes absent. The empty nerve-sheaths, both of the large and of the fine fibres, were recognised as rings, either round or distorted by pressure, their outlines being frequently marked in Marchi and osmium preparations by a marginal ring of fine black granules.

(*b*) In the spinal ganglia both ganglion cells and nerve fibres call for notice.

The cells, as indeed the ganglia themselves, appeared smaller than normal. Measurements through their longest diameters (when not perfectly round) gave their average diameters as 50μ, the limits between which they varied being from 25μ to 70μ. This is below the figure given by Lenhossék[23] for the average diameter of healthy cells of the larger sort, viz., 60 to 80μ. Striking was the amount of pigmentation they displayed. In very few was this limited to a mass lying between the nucleus and the thickened peripheral layer of cell plasma, or to a crescentic ring investing the nucleus. On the contrary, in the majority of the cells it occupied approximately two-thirds of the area of the cell plasma, obscuring the nucleus completely. It consisted of coarse pigment granules, insusceptible to stain, and agglomerated in many cases into dense clumps in which no differentiation of separate granules could be made out.

The cell plasma was invariably opaque and turbid, never in the least degree brightly refractile or translucent. It stained readily with plasma stain and then showed itself finely granular in structure. The finer details of the granules could not be made out owing to shortcomings in the staining, due to my want of familiarity with Mann's method. No vacuolation was present, but the normal marginal space between cell plasma and capsule figured and described by Lenhossék was frequently present. The nuclei were occasionally well defined as also were the nucleoli, but not in every cell. The cell processes also could only seldom be distinguished. The epithelium of the capsules appeared normal.

I am unable to venture on an opinion as to how far these appearances are abnormal or the expression of artefacts. The latter explanation readily suggests itself in connection with the shrinking in size. But, on the other hand, the method of hardening used by Lenhossék, from whom the normal measurements are taken, viz., corrosive sublimate and alcohol, would cause as much shrinking as the Müller's fluid and alcohol used in my own preparation. The amount of pigmentation that may be considered normal is another vexed question. Wollenberg, who discusses the matter,[22] apparently concludes, with Obersteiner, that the decision as to what is and what is not pathological is frequently an arbitrary one. The opacity of the cell plasma may —according to Trzebinski (quoted by Wollenberg, *loc. cit.*)—be produced by the Müller's fluid and alcohol employed. I can speak definitely only as regards vacuolation and fatty degeneration of the plasma, neither of which could be recognised in these sections.

On the whole, a comparison with the few normal specimens I possess leads me to the opinion that the ganglion cells were smaller and more pigmented than average normal cells. Whether this, however, corresponds with the pigmentary atrophy described by other observers, I am unable to say.

In the fasciculi of transversely divided nerve-fibres which appeared in the sections of the ganglia, it was easy to recognise marked degeneration. Numerous lacunæ were present, corresponding to spaces vacated by swollen medullary sheaths that had disappeared in handling. Many of the fibres showed granular and disintegrated myelin, and from many the axis cylinders had disappeared. Between these were spaces occupied by connective tissue, fine fibres, some apparently healthy, some undergoing degeneration, and empty Schwann's sheaths.

Logwood preparations showed a distinct multiplication of connective tissue nuclei, both in the endoneurium between the nerve fibres and in the interstitial tissue between the ganglion cells.

(*c*) *Below the Ganglia.*—The roots showed the same condition of degeneration as already described.

(*d*) *The Mixed Trunks.*—Tranverse sections here showed the two roots lying within a connective tissue-sheath apparently much too large for its contents.

The constituent portions of the nerve were easily recognised, in Pal-Weigert and Schäfer preparations, the anterior root showing, for the most part, regular and well stained fibres of uniform size, while the posterior showed a few dark stained discs and rings in the midst of a mass of unstained sclerotic tissue.

The Anterior Roots.—These consisted, for the most part, of large nerve tubes, of 10-12μ in diameter, with healthy sheaths and axis cylinders. They were not wholly free, however, from degenerative change. This affected chiefly the myelin sheath.

In some the myelin had separated from the Schwann's membrane and appeared loose in the interior of the tube. In others it was aggregated into a mass lying against the inner wall of the sheath. In others, again, the myelin appeared to have become absorbed, leaving a naked axis cylinder inside the tube so formed. This condition, well shown in benzo-purpurin and Mallory-hæmatoxylin preparations, appeared to resemble the denudation of the axis cylinder described by Babinsky and others as occurring in multiple sclerosis. A few fine fibres in good preservation were present in the anterior roots, but in much smaller numbers than in the posterior.

The Peripheral Nerves.

Those examined were the sciatic in the upper part of the thigh, the anterior crural, anterior tibial, internal plantar, median, ulnar, radial and posterior interosseous. Each and all of these showed extreme degeneration. By Schäfer's method not only the healthy medullated sheaths, but those also which contained degenerate but unabsorbed myelin, gave the black reaction. The unstained areas, therefore, corresponded to parts which were totally devoid of myelin, either healthy or degenerate.

To obtain a comparative estimate of the degrees of this degeneration various methods were adopted.

A modification of Sakaky's method[26] was used for estimating the number of healthy fibres in the fasciculi. By means of a mm. stage micrometer and a corresponding ruled ocular the number of healthy fibres present in a series of squares each with a side measurement of ·1 mm. was counted, and an average was taken from nine or sixteen of these squares. Observations on healthy nerve fibres gave 32 nerve fibres of 10 to 12_μ each in diameter as the average normal contents of such a square. With the normal figure of 32 was compared the number of healthy fibres per ·1 mm. square in the best preserved portion of the cross section of the nerve. The small fibres, of which large numbers were apparent in all of the peripheral nerves, were excluded from the calculation on account of the difficulty of getting any satisfactory estimate of their number in normal nerves.

As regards the larger fibres, it was found that in the majority of the nerves examined, viz., in the median, radial, posterior interosseous, sciatic, anterior crural, and internal plantar, occasional fields could still be found in which a few squares reached or approximated to the normal content of fibres.

In the ulnar and anterior tibial, however, in not a single field in the cross section of the nerve could an area containing 32 healthy fibres per square ·1 mm. be found.

The relative degree of sclerosis was roughly estimated by measuring the largest superficial areas in each nerve in which not a single healthy medullated fibre could be found.

The results may be thus tabulated :—

In the posterior interosseous nerve a whole field measuring ·3 mm. in diameter	contained not a single healthy fibre.
In the ulnar, a space measuring ·3 × ·15 mm.	,, ,, ,, ,, ,,
In the radial, a space measuring ·2 × ·2 mm.	,, ,, ,, ,, ,,
In the median, a space measuring ·18 × ·18 mm.	,, ,, ,, ,, ,,
In the anterior tibial, a space measuring ·175 × ·175 mm.	,, ,, ,, ,, ,,
In the internal plantar, a space measuring ·125 × ·15 mm.	,, ,, ,, ,, ,,

In the sciatic and anterior crural nerves no sclerotic area larger than ·1 mm. square void of healthy fibres could be found.

Contrary to one's expectation, it thus appeared that the greatest destruction of nerve fibres had taken place in the mixed nerves of the upper limb. In the nerves of the lower limb it appeared that portions more peripherally placed suffered greater degeneration than those nearer the cord. Connective tissue hyperplasia was extreme in those fibres in which the sclerosis was most marked, and reached its maximum in the internal plantar. In the same nerve the accompanying artery showed an extreme thickening of the intima, the lumen of the vessel being almost obliterated thereby. In the sciatic and anterior crural there was a great deposit of fat in the interfasicular connective tissue.

The degeneration in every case was of the ordinary parenchymatous type, the medullary sheaths being either granular, broken into clumps, disintegrated, with formation of droplets, or absorbed. Persistence of the axis-cylinders, such as was observed in the anterior roots, was nowhere seen in the peripheral nerves.

The Medulla.

Sections were cut from the whole length of the medulla with the exception of the region of the pyramidal crossing, which was unfortunately destroyed in removing the parts. The changes met with may be sufficiently described at three levels.

(*a*) Between the pyramidal decussation and the lower level of the olives.

The longitudinal fibres at this level comprise the funiculi graciles and cuneati, the lateral and pyramidal columns, and the two narrow layers which, lying on each side of the middle line dorsal to the anterior pyramids, represent the fibres of the inter-olivary layer or fillet.

It is these latter fibres and the adjoining tracts of the antero-lateral ground fibres which, alone of the longitudinal bundles, present healthy fields of well-stained fibres at this level. Of the remainder, the funiculus gracilis (Goll's column) maintains its former condition of almost complete sclerosis. The funiculus cuneatus (Burdach's column), better preserved than the preceding, shows a slender band of stained fibres along the margin which adjoins the central grey matter. In the lateral columns there is a general lightening of the stain, amounting in the region of the D. C. tract to complete sclerosis. Gower's tract shows here more definite signs of degeneration than were apparent in the cord. In the pyramids, there is a general faintness of the medullary stain, and, as was to be expected from the condition of the direct and crossed pyramidal tracts, numerous degenerated fibres are present.

Of the grey matter, the clavate nucleus is practically absent, being represented only by a very few rounded nerve cells.

The cuneate nucleus is also poorly developed, being considerably below its normal bulk. The caput cornu posterioris, however, now swelling into the tubercle of Rolando, appears to be recovering from its former atrophy, and shows a well-developed head of grey matter and numerous small, nucleated cells. The central canal appears normal. The ependymal lining is distinctly columnar, and outside the epithelial layer is a loosely-arranged gleial network, with numerous Deiter's cells.

The healthy and well-developed condition of the fillet layers already referred to occasioned some surprise, on account of the meagre development of the innermost strands of the internal arcuate fibres. These latter, proceeding from the nuclei of the posterior columns, bend round the central grey substance, and after decussating give rise to the fibres of the fillet layers. Owing probably to the sclerosis present in the clavate nucleus and the poor development of the cuneate nucleus, the strands passing from these nuclei to the fillet decussation are less full and less numerous than in a normal medulla. Some bundles of well-stained arcuate fibres which are present arise from the neighbourhood of the posterior horn, and occupy the outermost division of the internal arcuate fibres.

(*b*) Lower level of the inferior olive.

The healthy fibres of the fillet layers now appear as the well-stained interolivary layer, and, dorsally to these, the median area of the reticular formation is occupied by well-stained fibres, which, according to Ferrier,[43] are contributed from the anterior root-zone of the cord. The lateral area of the reticular formation is less well preserved. Towards the dorsal margin of the lateral column there is a considerable accession of healthy, dark-stained fibres apparent in cross section, and these are evidently related to the dark-stained bundles of internal arcuate fibres which are seen to proceed to the region of the corpora restiformia from the olives and the fillet.

The clavate nucleus is still absent as such from the postero-internal column, but traces of grey matter with infrequent round cells can be made out in its site.

The caudate nucleus is now well developed, and contains numerous nerve cells.

The olivary bodies are well developed, and the numerous fibres passing from them show the black stain of normal myelin.

The central grey matter and the posterior longitudinal fasiculus also appear normal.

The pyramids, however, are smaller than normal, and still contain many degenerated fibres.

(*c*) Upper level of olive (at and above calamus scriptorius).

The prominent feature from this level upwards is the gradual increase in the healthy fibres of the corpus restiforme of each side by the continued accession of arcuate fibres from the olivary bodies.

There still appears a small area of sclerosis in the site occupied by the direct cerebellar fibres. The pyramids continue of smaller bulk than normal, but they exhibit less evident signs of degeneration than at lower levels.

The cranial nuclei (XII. and X.) appear to be normal. The fibres of the fillet and of the posterior longitudinal fasiculus are well stained and healthy.

From this level upwards the medulla and pons exhibit no marked morbid change.

Cerebellum.

No signs of degeneration or maldevelopment could be found in the cerebellum. The inferior peduncle showed healthy fibres and axis-cylinders throughout. The plicæ showed a normal arrangement, with a well-marked nuclear layer, and numerous healthy Purkinje's cells.

REMARKS ON THE CASE.

In its clinical features and its post-mortem appearances, the case reported shows no peculiarity. With the former I do not intend to deal, as the matter has been so recently and exhaustively treated by Mackie Whyte in this Journal.[42]

None of the pathological conditions described are new to the literature of the disease, with the trivial exception, perhaps, of the abnormal arrangements described as occurring in the posterior column of the cord, in the upper dorsal and mid-cervical regions.

In many features the case bears a close resemblance to some of the more recently reported cases, *e.g.*, those of Simon (16, in summary of autopsies), Bonnus (*ib.* 17), Mirto (*ib.* 13), and Guizetti (*ib.* 12). In four of these, viz., 12, 16, 17, and my own, the degeneration of the posterior column reached its maximum in the dorsal region. In the same four cases the cornu-commissural tract, and fibres representing Flechsig's oval tract, escaped injury either relatively or entirely.

The degeneration of the crossed pyramidal and direct cerebellar tracts, recorded in nearly every reported case, was present in these cases too. In addition Lissauer's tract was more or less affected in each, as it was also in cases 11 and 10.

In all, as indeed in every recorded case where they have been examined, the posterior roots were sclerosed. The condition of the spinal ganglia is not mentioned in cases 16 and 17. In 12 and 13, however, as also in Blocq and Marinesco's case, 11, and probably also in my own, 18, changes were present. Interstitial hyperplasia, with changes in the ganglion cells, vacuolation, or shrinking, are mentioned in every case.

In the medulla, sclerosis of the posterior columns, with degeneration of nuclei, is recorded in cases 12, 16, 17, 18, and also in 11, and degeneration, in the bulb, of the direct cerebellar fibres, is also recorded in cases 11, 12, 16 and 18.

Similar changes in the posterior columns of the medulla are reported by Friedreich (cases 2 and 4), and were also observed in the doubtful cases of Brousse and Tedeschi.

Some degeneration of the fibres of the anterior pyramids occurred in Guizetti's case and my own.

The cerebellum has been found normal in every case in which its condition has been recorded, with the exception of Auscher's doubtful case.

The peripheral nerves were atrophied or degenerated in cases 12, 13, 17 and 18, severely so in the last case.

In the light of these cases, together with the fact that similar changes have been recorded by Friedreich and by Rutimeyer, it must be admitted that degeneration of the peripheral nerves, which may reach an extreme degree, ought in future to find mention in any complete account of the morbid anatomy of Friedreich's disease.

The nature of the fine fibres observed in the peripheral nerves in this as in other cases of Friedreich's disease is a question of great interest.

Auscher[10] describes those present in his case as fibres having embryonal characters, *i.e.*, with an axis-cylinder, and either a very slight myelin sheath or none at all. He expressly states that he found no degenerated tubes nor empty sheaths.

Guizetti[11] records the presence of numerous fine fibres with a diminution in the number of coarse fibres. He describes and figures various histological conditions of the fine fibres in the nerves and the posterior roots, viz., some—a majority—with axis cylinders, some without, some apparently deprived of medullary sheaths, and others in which the sheaths stained slightly with osmic acid. He assumes these conditions to be the result of atrophy.

In my own case several varieties of fine fibres could be distinguished, viz. :—

(*a*) Fine fibres with a slender axis-cylinder and an apparently medullated sheath. I say *apparently* because the substance of the sheath, though colouring with plasma stains, and very faintly also with chromatin stains, yet failed to take up the specific myelin stains, such as those of Weigert and Schäfer. This fact has been noted also by Tedeschi.[15]

(*b*) Fine fibres showing more or less traces of degeneration, *e.g.*, (*a*) a granular condition of the sheath, (β) hypertrophy of the axis cylinder, either with or without a thin investment of myelin, or (γ) disappearance of the axis cylinder.

(*c*) Empty fibres, myelin and axis-cylinder having undergone absorption, the Schwann's sheaths remaining having frequently fine granules, staining black with osmium, attached to their inner surfaces.

All these appearances have been noted and described in the degeneration of peripheral nerves which accompanies various scleroses of the cord. Westphal figures them in a case of postero-lateral sclerosis (*Archiv. für Psychiat.*, Bd. viii., taf. xi.), and again in a case of central and peripheral degeneration due, he thinks, to tubercular infection (*ib.*, Bd. xvi., taf. xi.). Sakaky,[26] and Oppenheim and Siemerling,[28] describe them in the peripheral neuritis accompanying cases of tabes. Many observers have described the arrangements of the fine fibres as being in closely packed clusters or fasciculi, as if held together by some outer investment. Oppenheim and Siemerling (*loc. cit.*) describe bundles of well-preserved rods of fine calibre inside the empty Schwann's sheaths of larger fibres. These observations suggest the presence of fibrils of regeneration rather than that of atrophied fibres.

Since Déjérine has shown[29] that the peripheral degeneration in some cases of tabes attacks first the distal extremity of the peripheral neuron, and may co-exist with complete integrity of the spinal ganglion and posterior spinal root, it is reasonable to suppose that the peripheral degeneration in cases of sclerosis of the cord, such as those alluded to above, may be akin to that which occurs in the toxæmic states due to lead, alcohol, diphtheria, &c. In these, as Gombault,[37] S. Mayer,[38] Pitres and Vaillard,[39] Gudden,[24] and others have described, there occurs, after the degenerative period, an outbudding of small regenerative fibres (" segments intercalaires," "schaltstucke"), which, in cross-section, would present the characters of the fine fibres described under (*a*). Indeed, it seems difficult to account in other ways for the presence of these fine medullated and cylindered fibres in the midst of otherwise general degeneration. If Fleming's view[31] be correct, that the normal fine fibres in peripheral nerves are the first to undergo degenerative changes, the fine fibres under discussion can hardly represent the original fine fibres present normally in every peripheral nerve, and unless simple atrophy of nerve fibres can take place without a trace of molecular change or disintegration, it is equally difficult to conceive these fine fibres as originating in the atrophy of the larger ones. It seems, at least, equally probable that they may be single or multiple regenerative fibres, the outcome of the segmental neuritis of larger tubes. Further, there is no cause hindering the degeneration in their turn of the fine regenerative fibres. As Gudden remarks[24] (p. 731):—"One factor only need be assumed, viz., that the patient is a debilitated individual, and that the toxic virus does not at once escape from the system. In such a case the restitution, in the first place, of the nerve fibres, proceeds slowly, and secondly, these latter, when scarcely in working order, either through the feebleness of the organism or through the presence of some fresh virus. . . . are checked in their further development and again fall into decay." Gudden instances as an illustration of "fresh virus" the infection of tubercle, and it is noteworthy that if the doubtful cases of Friedreich's disease in the appended list be included, a history of tubercle is present in seven out of the twenty-two autopsies recorded.

Such a process as is suggested above would well account for the appearances found in the fine fibres in classes (*b*) and (*c*). It may be objected to this explanation of the presence of the fine fibres that the primary pathological processes, central and peripheral, in Friedreich's disease, have not been shown to depend on any toxæmic dyscrasia, inherited or acquired. The same objection, however, applies equally to amyotrophic lateral sclerosis, and to the hereditary form of muscular atrophy. These diseases affect the peripheral motor neurons, much, it appears to me, as Friedreich's disease affects the peripheral sensory neurons associated with muscle. In both these forms of muscular atrophy Gombault has observed [37] in the degenerate peripheral nerves the occurrence of segmental neuritis with regenerative processes.

The extreme preponderance of the degeneration in the posterior roots rather than the anterior indicates that it is the afferent fibres of the peripheral nerves that are chiefly attacked. The fact, moreover, frequently recorded, that the patients' sensibility to touch, pain, and temperature remains unimpaired throughout, suggests that it is the afferent nerves from muscles that chiefly suffer. This agrees with Guizetti's conclusion, who was of opinion that in his own case there existed a systematic atrophy of sensory fibres. In his case, however, he considered that muscular and cutaneous fibres were both affected, and it should be remembered that, clinically, the patient's tactile and thermic sensibility were diminished below the knees, and that some tactile impairment was present in the hands.

The presence of abnormal arrangements in the structure of the cord which I have described as occurring in the dorsal and cervical portions is suggestive of structural anomaly dating back to the developmental period of the cord. Sections containing these abnormalities were variously stained in the endeavour to ascertain the finer structure of the growths. The nerve fibres traversing the areas were invariably degenerated, and none of them possessed axis cylinders. The intervening ground substance resembled closely the sclerotic tissue present in the posterior parts of Goll's column, and its density opposed a serious obstacle to its examination. The felt-like tissue composing it shrank extremely in the process of dehydrating and clearing, so that thin sections were destroyed by the tearing consequent on the shrinkage. Deiter's cells, however, could not be distinctly recognised, but corpora amylacea were present in considerable numbers. The staining reactions were those of glial tissue, and in all probability the structure was that of a circumscribed gliomatous mass of long standing, in which the cellular elements seemed to have disappeared.

Finally, no appearance at all corresponding to Déjérine's description of whorls of glial tissue could be recognised in this case. The grey sclerotic areas in the posterior and lateral columns were found histologically to have the usual structure of neuroglial hyperplasia elsewhere. Numerous large Deiter's cells were present, frequently of elongated or lanceolate shape, as if obliquely-lying elongated cells had been divided in a plane approximating that of their longest diameter. In all, or nearly all, the faintly stained granular nucleus could be recognised, and in some the processes could be traced for a short distance from the cell. These elongated cells and smaller rounded ones formed dark-stained masses in a dense spongy-looking groundwork, the minute dark points in which probably corresponded to gleial fibres in cross-section. The cells were least distinct, and stained less freely in the older and more completely sclerosed areas, while they stained most distinctly in the transition zones between healthy and degenerating tissue. Where they stained least distinctly, *e.g.*, in the peripheral portion of the posterior columns, there numerous corpora amylacea were thickly strewn, and the converse of this arrangement held equally true, viz., the corpora amylacea were least numerous in those regions where the glial cells were most distinct. The same histological features characterised the areas of degeneration in the various columns of the cord. No special peculiarities could be made out in the lateral areas of sclerosis. The vessels and capillaries were nowhere markedly thickened, but were everywhere distended with blood cells.

The last mentioned fact is of interest in relation to the vascular theory of the causation of the disease which was favoured by Pitt, and by Blocq and Marinesco. These observers found changes in the blood vessels of the posterior columns, as also did Schultze in his second case, and Everett Smith.

On the other hand such changes were absent in the cases of Rutimeyer, Déjérine and Letulle, Guizetti, and in my own.

Guizetti, who discusses this matter, points out that disease of the posterior vascular system of the cord would leave unexplained the condition of the posterior roots and spinal ganglia, and that Clarke's column, which is invariably sclerosed, is supplied by the anterior system of vessels. Moreover, that such vascular dilatations as Pitt and Blocq and Marinesco have recorded, are a frequent accompaniment of sclerosis of the cord, and probably secondary to it.

On the whole it appears that our present knowledge of the pathogenesis of the disease cannot be better summed up than in Guizetti's conclusion, that Friedreich's disease depends upon a congenital predisposition, in consequence of which, in the early years of life, certain systems of fibres and nerve cells undergo a process of progressive atrophy, and that the process is independent of any contributory effect from vascular alterations.

To Guizetti's conclusion might be added that by "certain systems of fibres and cells" ought perhaps to be understood the following :—

(*A*) (Due to arrested development at the eighth month?) Tracts in the cord which are the latest to undergo medullation, viz., the pyramidal tracts, direct and crossed, and the postero-internal tract.

(*B*) (Secondary to mal-development of Goll's column?) Systems of fibres and cells functionally related to the postero-internal tract. The extent of the atrophic process as to systems attacked, and degree of degeneration in individual systems, may be dependent upon the duration of the disease. These systems may include :—

(*a*) The peripheral sensory neuron complex in its entirety, viz., peripheral sensory fibres, ganglion cells, posterior roots and root-zones (Burdach's, Lissauer's), fibres to Clarke's columns, to the middle zone, and to the anterior cornua.

(*b*) Portions of the central sensory neurons, viz., cells in posterior horns, with associated ascending fibres, cells in Clarke's columns, with associated fibres (direct cerebellar tract), cells in middle zone, with associated fibres (Gower's tract), cells in medullary nuclei, clavate and cuneate, with associated fibres (internal arcuate).

The atrophic changes in every system are arrested in the medulla, when the level of the lower olive is reached.

I append a summary in tabular form of the pathological conditions present in the autopsies of Friedreich's disease to which I have been able to refer, and I believe the list is fairly complete up to the date of its compilation (July, 1898). A useful summary of most of the cases included was given by Tedeschi [15] in 1896. I have excluded from the list all cases of the cerebellar type of the disease with which Nonne, Menzel, Marie, Sanger Brown and others have made us familiar, because it is clear—*pace* Senator—that these form a group quite distinct, clinically as well as pathologically, from the type described by Friedreich.

I have also placed in a separate list the doubtful cases of Kahler and Pick, Brousse, Auscher, and Tedeschi.

Kahler and Pick's case presents some close resemblances to hereditary ataxia. The age at onset—on menstruation, at 16—the general course of symptoms, the loss of knee-jerk, the impaired speech, the preserved pupil reflex, and the ataxia, all agree with Friedreich's symptom-complex. In addition spinal curvature was present, and a sister of the patient is said to have been affected similarly to herself.

On the other hand, Westphal *(Archiv. für Psychiat.*, Bd. ix., p. 697), draws attention to the early onset of paralytic weakness in the case, with outstretched legs and pointed toes, as dissociating it from Friedreich's group. In addition, nystagmus was absent, and there is no mention of choreiform movements, or of pes cavus.

Very similar to Kahler and Pick's case is that of Erlicki and Rybalkin [40] affecting also a young girl soon after puberty. In this case the rude strength of the legs was well maintained during the early period, and the majority of the symptoms of Friedreich's disease were present. On the other hand, deformity of the feet and spine were absent, as also were nystagmus and speech affection. More important, perhaps, was the hurried course of the disease, the symptoms developing with undue rapidity, and death ensuing from tubercular diarrhœa in twenty months from the onset. For this reason I have omitted the case.

Brousse's and Auscher's cases both concern women of later age, 24 years and 24½ respectively. In the former neither spinal nor foot deformity was present, nor, apparently, nystagmus. In the latter the reflex to light was absent, and post-mortem, brain and cerebellum were found of small size, while the lateral columns of the cord were unaffected.

In Tedeschi's case, as Mackie Whyte has pointed out,[42] many important clinical details are unfortunately omitted, *e.g.*, the condition of the knee-jerks, state of the spine, &c. The onset of the symptoms was anomalous, the arms being attacked before the legs, the gait was tabetic in character rather than cerebellar, and the patient was insane.

A case reported by Mitchell Clarke[41] has also been omitted because the complication of cerebellar tumour which was present vitiates the pathological record for purposes of comparison.

It only remains for me to express my grateful acknowledgments to Sir T. Grainger Stewart, who suggested to me the examination of the peripheral nerves, and to Dr. Eurich of Bradford, for kind assistance in the technic of neuroglia staining.

SUMMARY OF AUTOPSIES ON UNDOUBTED CASES.

Case 1.

A.—*Name and Duration.*—Andreas Lotsch; 15 years ill. Died at 38; cause, typhoid.

B.—*Author and Reference.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii., s. 1, 1863.

C.—*Cord.*—Small.

D.—*Membranes.*—Dural sac contained fluid. Pia milky and thickened, soft adhesions to cord.

E.—*Posterior Column.*—Grey, translucent, firm, from above lumbar swelling to calamus. Sclerosis most marked above lumbar swelling. *Microscopically* —Atrophy of nerve tubes and presence in their place of a finely fibrillar connective tissue—made up, in part, of the shrunken Schwann's sheaths. Between these elements a finely granular ground substance, clearing with acetic acid, and then revealing many round and oval nuclei.

F.—*Lateral Column.*—Normal.

G.—*Anterior Column.*—Normal.

H.—*Posterior Horn.*—Normal.

J.—*Anterior Horn.*—Normal.

K.—*Clarke's Column.*—Normal.

L.—*Spinal Roots.*—Posterior roots atrophied.

M.—*Spinal Ganglia.*—No mention.

N.—*Peripheral Nerves.*—Some atrophy in sciatic. Less in nerves of cervical and brachial plexus. More intense degeneration in hypoglossi.

O.—*Central Canal.*—No mention.

P.—*Medulla.*—Corpora restiformia slightly attacked.

Q.—*Cerebellum.*—Normal.

R.—*Cerebrum.*—Normal.

S.—*Remarks.*—Walls of capillaries in affected areas show fatty globules.

Case 2.

A.—*Duration.*—Justinie Suss; 15 years ill. Died of typhoid at 31.

B.—*Author.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii. s. 1, 1863.

C.—*Cord.*—Small.

D.—*Memb.*—Pia thickened and adherent to dura and to cord.

E.—*P. Col.*—Sclerosed between lumbar and cervical swellings. Above the cervical swelling only a thin zone, next periphery affected. Postero-median fissure obliterated.

F.—*L. Col.*—Sclerosed between lumbar and cervical swellings in the parts adjacent to the posterior column, but to a less extent than the posterior column.

G.—*A. Col.*—Normal.

H.—*P. Horn.* **J.**—*A. Horn.* **K.**—*Clarke's Col.*	Grey matter of cord sclerosed in dorsal region.

L.—*Sp. Roots.*—Posterior roots atrophied.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Interstitial hyperplasia in the nerve trunks of the extremities.

O.—*C. Canal.*—Occluded with small cells.

P.—*Med.*—Sclerosed in posterior column.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*

Case 3.

A.—*Duration.*—Salome Suss; duration 14 years. Died at 28 of typhoid.

B.—*Author.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii., s. 1, 1863.

C.—*Cord.*—As in former cases.

D.—*Memb.*—Pia thickened and opaque, and adherent to dura.

E.—*P. Col.*—Sclerosed throughout, most marked in dorsal region.

F.—*L. Col.*—Sclerosed, in dorsal region, in posterior part of lateral column on left side.

G.—*A. Col.*

H.—*P. Horn.*

J.—*A. Horn.*

K.—*Clarke's Col.*

L.—*Sp. Roots.*—Posterior roots atrophied.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*—A double canal containing fluid in cervical region.

P.—*Med.*

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*

Case 4.

A.—*Duration.*—Fr. Suss; duration 27 years. Died at 42 of typhoid.

B.—*Author.*—Friedreich and Schultze. *Virchow's Archives*, Bd. lxx., s. 140.

C.—*Cord.*—As in former cases.

D.—*Memb.*—Pia thickened and opaque, and adherent to dura.

E.—*P. Col.*—Sclerosed throughout. In cervical region Goll's column chiefly attacked.

F.—*L. Col.*—Sclerosed, in posterior part, throughout whole length of cord. A small band of healthy tissue intervening between sclerosed tissue and posterior column.

G.—*A. Col.*—In the cervical region severe sclerosis of the right pyramidal tract, adjoining the anterior fissure. This extended upwards beyond the decussation, but did not extend downwards into dorsal region.

H.—*P. Horn.*—Atrophied.

J.—*A. Horn.*—Atrophy of grey substance at cervical swelling. Ganglion cells fewer and smaller than normal.

K.—*Clarke's Col.*—Cells atrophied.

L.—*Sp. Roots.*—Posterior roots atrophied.

M.—*Sp. Ganglia.*—Normal.

N.—*Peri. Nerves.*—Normal.

O.—*C. Canal.*

P.—*Med.*—Small. Sclerosed in Goll's and Burdach's columns, with atrophy of the ganglion cells of both nuclei. Anterior pyramids normal. Connective tissue hyperplasia, with corpora amylacea, in corpora restiformia as high as the level of cal. script.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Walls of vessels thickened, especially in neighbourhood of posterior column.

Case 5.

A.—*Duration.*—Charlotte Lotsch; duration 35 years.

B.—*Author.*—Schultze. *Virchow's Archives*, Bd. lxxix., s. 132.

C.—*Cord.*—Thin and flattened.

D.—*Memb.*—Pia thickened and opaque.

E.—*P. Col.*—Sclerosed. In the cervical region a strip of less degenerate substance, 1 mm. broad, remains, dorsal to the posterior commissure. Postero-external fields also contain some normal fibres.

F.—*L. Col.*—Posterior parts sclerosed up to the periphery, but the degeneration did not extend so far forward as in case 4.

G.—*A. Col.*—Both sides sclerosed for some distance from decussation downwards, in a comma-shaped tract.

H.—*P. Horn.*—No mention.

J.—*A. Horn.*—No mention.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed.

M.—*Sp. Ganglia.*—No abnormality.

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*—Normal, except for some hypertrophy of connective tissue.

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*

Case 6.

A.—*Duration.*—Clara S.

B.—*Author.*—Everett Smith. *Boston Medical and Surgical Journal,* October, 1885.

C.—*Cord.*—Asymmetrical, small.

D.—*Memb.*—Meninges injected and adherent to bone.

E.—*P. Col.*—Sclerosed ; with exception of a small area lying dorsal to the posterior commissure.

F.—*L. Col.*—Sclerosis of crossed pyramidal tract.

G.—*A. Col.*—Sclerosis of direct pyramidal tract, bordering on anterior fissure, but less intense than in the posterior columns.

H.—*P. Horn.* }
J.—*A. Horn.* } Nerve cells less numerous than normal, and much altered.

K.—*Clarke's Col.*—No mention.

L.—*Sp. Roots.*—Posterior sclerosed.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—Smith thinks the disease due to arrested development of some of the cells and fibres of the cord.

Case 7.

A.—*Duration.*—R. S. ; duration 14 years. Died at 29 of cardiac dilatation and failure.

B.—*Author.*—Newton Pitt. *Guy's Hospital Reports,* 1887, p. 369.

C.—*Cord.*—Unusually small.

D.—*Memb.*—Unaffected.

E.—*P. Col.*—Sclerosed. Goll's column throughout. Burdach's column severely so in posterior part. Escape of a narrow strip bounding posterior horn and root, especially along anterior half.

F.—*L. Col.*—Sclerosis of crossed pyramidal and cerebellar tracts. Most marked in dorsal region ; less so above. A strip of marginal sclerosis extends forward, anterior to the direct cerebellar tract (Gower's tract ?).

G.—*A. Col.*—Periphery slightly sclerosed above, extending, in lower cervical region, to antero-median fissure.

H.—*P. Horn.*—In some sections sclerosed.

J.—*A. Horn.*—Unaffected.

K.—*Clarke's Col.*—Sclerosed in some sections.

L.—*Sp. Roots.*—Posterior sclerosed; anterior normal.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Radial and anterior crural showed no degeneration.

O.—*C. Canal.*—Double canal present at decussation of pyramids.

P.—*Med.*—Marked degeneration of funiculus gracilis; less severe of funiculus cuneatus. Some degeneration in funiculus rotundus.

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—Pitt associates the disease with a tendency to vascular degeneration and an ill-developed spinal cord.

Case 8.

A.—*Duration.*—H. Kern; duration 14 years. Died at 20.

B.—*Author.*—Rutimeyer. *Virchow's Archives*, Bd. cx., s. 215.

C.—*Cord.*—Small.

D.—*Memb.*—Pia thickened, numerous adhesions, and opaque.

E.—*P. Col.*—Sclerosed. Severe of Goll's column; less so of Burdach's. Escape of Lissauer's tract and of Flechsig's oval tract.

F.—*L. Col.*—Sclerosis of crossed pyramidal and direct cerebellar tracts, but less severe than that of posterior column. Escape of lateral limiting layer.

G.—*A. Col.*

H.—*P. Horn.* } Grey matter of cornua appears normal.
J.—*A. Horn.* }

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed, most markedly in dorsal region.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Marked degeneration of sciatic and median.

O.—*C. Canal.*

P.—*Med.*—Small. Sclerosis of funiculi graciles and cuneati.

Q.—*Cbl.*

R.—*Cbr.*—Normal.

S.—*Remarks.*

Case 9.

A.—*Duration.*—B. Kern; duration 9 years.

B.—*Author.*—Rutimeyer. *Virchow's Archives*, Bd. cx., s. 215.

C.—*Cord.*—Small.

D.—*Memb.*—No sub-dural exudation; pia thickened.

E.—*P. Col.*—Sclerosed; with exception, in cervical and lumbar regions, of small fields, as in former case.

F.—*L. Col.*—Sclerosis of crossed pyramidal and direct cerebellar tracts, with escape of lateral limiting layer. Some marginal degeneration extends forward beyond region of D. C. T. (Gower's tract?) Lateral sclerosis most marked in the cervical region.

G.—*A. Col.*

H.—*P. Horn.*

J.—*A. Horn.*

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*—Small.

Q.—*Cbl.*

R.—*Cbr.*—Normal.

S.—*Remarks.*—Rutimeyer considered the degeneration in both these cases to be identical with that occurring in tabes.

Case 10.

A.—*Duration.*—Paul Par.; duration 11 years. Died at 21, of mitral stenosis and pulmonary apoplexy.

B.—*Author.*—Déjérine and Letulle. *La Médecine Moderne*, April, 1890.

C.—*Cord.*—Small.

D.—*Memb.*—Adherent.

E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column. Marked sclerosis of Burdach's. Sclerosis of Lissauer's tract.

F.—*L. Col.*—Slight sclerosis of crossed pyramidal tract. Medium atrophy of direct cerebellar tract.

G.—*A. Col.*—Unaffected.

H.—*P. Horn.*—Shrunken in lumbar region.

J.—*A. Horn.*

K.—*Clarke's Col.*—Atrophy of fibres and cells.

L.—*Sp. Roots.*—Atrophy of posterior roots.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*—Peri-ependymal glioma.

P.—*Med.*

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.* — Déjérine described in this case whorls of neuroglial fibres, which in L. S. appeared as undulating intercrossing fibres of great length. While this was the condition of the sclerosed posterior columns, in the lateral column on the other hand the sclerosis was a vascular one, and was associated with thickening of the pial prolongations. The lateral sclerosis he considered secondary to a meningo-myelitis.

Case 11.

A.—*Duration.*—Suzanne Desch.; duration 9 years. Died at 19, of pulmonary phthisis.

B.—*Author.*—Blocq and Marinesco. *Archives de Neurologie*, May, 1890.

C.—*Cord.*—Small.

D.—*Memb.*—Normal.

E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column. Severe sclerosis of Burdach's column. Escape of Westphal's postero-marginal zone. Lissauer's tract affected below but intact above.

F.—*L. Col.*—Sclerosis of crossed pyramidal and of direct cerebellar tracts, most marked in dorsal region. In lateral column, dilatation of vessels, with symmetrically placed hæmorrhagic exudates. Thickened pial trabeculæ are present in the sclerosed area. Gower's tract unattacked.

G.—*A. Col.*—Unaffected.

H.—*P. Horn.*—Diminution in number of large transversal fibres. Ascending fibres less affected.

J.—*A. Horn.*

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Sclerosis of posterior roots. Most marked in lumbar region.

M.—*Sp. Ganglia.*—Attacked. Disappearance of fibres. Peripheral vacuolation of ganglion cells. Hypertrophy of connective tissue.

N.—*Peri. Nerves.*—Not examined.

O.—*C. Canal.*

P.—*Med.*—Loss of fibres from posterior pyramids, less marked in corpora restiformia. Anterior pyramids unaffected. Cerebellar tract degenerated. Above olive a cavity, apparently due to capillary dilatation. Destruction of fibres of raphe.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Blocq and Marinesco consider the lesions in all the columns affected to consist in atrophy of nerve fibres and thickening of neuroglia. The fibres present have no relation to the pial system. They consider the morbid change starts from blood vessels, but that there is also a developmental lesion.

Case 12.

A.—*Duration.*—A. G.; duration 18 years. Death at 28 from heart disease.

B.—*Author.*—Guizetti. *Riforma Medica*, June, 1893, and *Il Policlinico*, 1894, p. 438.

C.—*Cord.*—Small.

D.—*Memb.*

E.—*P. Col.*—Sclerosed; with exception of a tract posterior to commissure and adjacent to posterior cornua (cornu-commissural tract ?). Escape of Flechsig's "centrum ovale." Lissauer's tract degenerated in dorsal region. Degeneration less severe in cervical region, where there is improvement in Burdach's column.

F.—*L. Col.*—Many degenerated fibres in crossed pyramidal tract in lumbar and dorsal region. Fewer in cervical region. D. C. T., and Gower's tract attacked from dorsal region upwards. Escape of lateral limiting layer.

G.—*A. Col.*—Unaffected.

H.—*P. Horn.*—Atrophied, cells few and degenerated.

J.—*A. Horn.*—Normal.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots sclerosed. Anterior healthy.

M.—*Sp. Ganglia.*—Diminution in size of ganglion cells with progressive atrophy of some. Compensatory hyperplasia of connective tissue.

N.—*Peri. Nerves.*—Much atrophied. Many fine fibres present.

O.—*C. Canal.*—Pervious for a short distance only.

P.—*Med.*—Degeneration of Goll's and Burdach's columns. Horizontal fibres unaffected. Some degenerated fibres in anterior pyramids.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Guizetti found no alteration of pial septa, or of blood vessels. The fine fibres in the peripheral nerves proceed solely from the posterior roots, and are therefore sensory. The fine fibres in the muscular branches have the same origin. Guizetti concluded that in this case of Friedreich's disease there existed a systematic atrophy of sensory nerves, both cutaneous and muscular.

Case 13.

A.—*Duration.*—L. P.; duration 5 years. Death at 19 from tubercular pleurisy.

B.—*Author.*—Mirto. *Giornale del'Assoc. dei Medici e Naturalisti*, Anno iv., 1893.

C.—*Cord.*

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Goll's and Burdach's columns degenerated. Lissauer's tract attacked.

F.—*L. Col.*—Degeneration of crossed pyramidal tract, diminishing from below upwards. Slight degeneration of direct cerebellar tract, and of Gower's tract.

G.—*A. Col.*

H.—*P. Horn.*
J.—*A. Horn.* Thinning of nervous reticulum in anterior and posterior horns, with atrophy of cells. Most marked in dorsal region.

K.—*Clarke's Col.*—Extreme sclerosis.

L.—*Sp. Roots.*—Posteror roots sclerosed.

M.—*Sp. Ganglia.*—Atrophic degeneration of cells. Thinning of nervous reticulum. Hyperplasia of connective tissue.

N.—*Peri. Nerves.*—Motor nerves degenerated; sensory not examined.

O.—*C. Canal.*

P.—*Med.*

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The sympathetic ganglia were also examined and found normal.

Case 14.

A.—*Duration.*—Female; duration 18 years. Died at 28 from diabetes and tuberculosis.

B.—*Author.*—Burr. *University Medical Magazine*, Philadelphia, June, 1894.

C.—*Cord.*

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column; less severe of Burdach's; from lower lumbar to highest cervical region.

F.—*L. Col.*—Degeneration of C. P. T. from lumbar swelling to upper cervical region. Degeneration of D. C. T. from mid-dorsal to upper cervical.

G.—*A. Col.*—Degeneration of D. P. T. from mid-dorsal to upper cervical regions.

H.—*P. Horn.*—Slight affection of posterior horns. Degeneration of ganglion cells.

J.—*A. Horn.*—Atrophy of a few cells.

K.—*Clarke's Col.*—Marked degeneration.

L.—*Sp. Roots.*—Posterior sclerosed; anterior intact.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—The reporter adheres to Dejerine and Letulle's theory of the nature of the changes in the posterior and lateral columns.

Case 15.

By an oversight the following was omitted from the Description of the Cord in Mirto's case, No. 13 in Summary:—

Cord.—Small.

C. Canal.—Occluded.

Medulla.—Goll's and Burdach's columns degenerated as far as their nuclei. Integrity of cellular elements of nuclei. Band of Reil (*scleife*) healthy. Degeneration of D. C. T. could not be traced beyond pyramidal decussation. Pyramidal fibres normal at, and above decussation.

O.—*C. Canal.*

P.—*Med.*—Degeneration "probably" extended into medulla.

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—For clinical history see *Journal of Mental and Nervous Diseases*, vol. xv., 1890. Numerous holes (? perivascular spaces) ·5 to 2 mm. in size were scattered through grey and white matter of the cord. Presence of gliosis in sclerosed area could not be affirmed.

Case 16.

A.—*Duration.*—Ad. Rouzier; duration 5 years. Death at 17 from unexplained cause, not revealed on autopsy

B.—*Author.*—Simon. *Le Progrés Médical*, September, 1897.

C.—*Cord.*—Small.

D.—*Memb.*—Thickening of soft membranes on posterior aspect of cord.

E.—*P. Col.*—Sclerosed. Most marked in dorsal region, but almost as severely in cervical, too. Lissauer's tract attacked in sacral region, but cornu-commissural and septo-marginal fibres escape. In lumbar region, Flechsig's centrum ovale is attacked, but cornu-commissural fibres again escape.

F.—*L. Col.*—Sclerosed, but less so than posterior column. The C. P. T. and D. C. T. equally affected from the upper lumbar region upwards. The lateral limiting layer escapes.

G.—*A. Col.*—Direct pyramidal tract affected from upper dorsal region upwards.

H.—*P. Horn.*—Myelinic reticulum degenerated. Diminution in number and in volume of ganglion cells.

J.—*A. Horn.*—Diminution in number and volume of ganglion cells, but the motor cells of front part of the cornua are well developed.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots sclerosed throughout; more so in lumbar and dorsal regions, less so in cervical.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*—Sclerosis of cuneate nucleus. More severe sclerosis of funiculus gracilis. Cerebellar bundle attacked.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The neuroglial tissue in the sclerosed areas is in some places arranged in perivascular whorls. Vessels but slightly altered. No marked proliferation of interstitial nuclei.

Case 17.

A.—*Duration.*—Jules G.; duration 13 years. Died at 39 of pleurisy (tubercular).

B.—*Author.*—Bonnus. *Nouvelle Iconographie de la Salpêtrière*, No. 3, 1898.

C.—*Cord.*—Small.

D.—*Memb.*—Healthy.

E.—*P. Col.*—Sclerosed. Most marked in dorsal region. Posterior and median radicular zones most attacked. Anterior radicular only affected above dorsal region. Lissauer's tract attacked in sacral and lumbar regions; less so in dorsal. Cornu-commissural tract and centrum ovale relatively, but not absolutely, preserved.

F.—*L. Col.*—Sclerosed, but less so than the posterior column. C. P. T. attacked throughout. D. C. T. attacked at and above dorsal region. Gower's tract also attacked.

G.—*A. Col.*—Direct pyramidal tract sclerosed in dorsal region, and still more affected in cervical region.

H.—*P. Horn.*

J.—*A. Horn.*

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots much degenerated.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Median, sciatic, anterior tibial, and musculo-cutaneous degenerated.

O.—*C. Canal.*

P.—*Med.*—Goll's and Burdach's nuclei attacked; otherwise normal.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Bonnus's case was diagnosed as Friedreich's disease by Charcot. It was characterised by lightning pains, which have been recorded in Friedreich's disease by Charcot and Dejerine.

Case 18.

A.—*Duration.*—Henry W.; duration 24 years. Died at 30, of heart failure during measles.

B.—*Author.*—Mackay.

C.—*Cord.*—Small.

D.—*Memb.*—Healthy; some thickening of dura.

E.—*P. Col.*—Sclerosed. Maximum degree in upper dorsal. Least degree in lowest lumbar, where cornu-commissural tract escapes relatively. Some fibres of septo-marginal tract escape in mid-lumbar. Lissauer's tract sclerosed, but gradual improvement in same from upper dorsal region upwards. Posterior root zones improve *pari passu* with Lissauer's tract.

F.—*L. Col.*—Sclerosed. C. P. T. from lumbar enlargement to upper cervical. D. C. T. from lower dorsal to calamus scriptorius. Maximum degeneration in mid-dorsal region. Lateral limiting layer intact throughout. Gower's tract slightly attacked in cervical region.

G.—*A. Col.*—Direct pyramidal tracts on both sides sclerosed from lower dorsal upwards. Maximum in cervical region.

H.—*P. Horn.*—Sclerosed both in longitudinal and transverse fibres. Ganglion cells for most part absent. Fibrillar plexus poor in myelin.

J.—*A. Horn.*—Normal, except for some slight diminution in volume of motor cells.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed, but contain many fine fibres. Anterior show slight degenerative change.

M.—*Sp. Ganglia.*—Shrinkage and extreme pigmentation of ganglion cells. Increase of interstitial connective tissue. Degeneration of ascending fibres passing through ganglia.

N.—*Peri. Nerves.*—Sciatic, anterior crural, anterior tibial, internal plantar, median ulnar, radial and posterior interosseous, all extremely degenerated.

O.—*C. Canal.*—Occluded by round cells in most of its course. Normal above decussation of pyramids.

P.—*Med.*—Sclerosis of cerebellar tract. Sclerosis of funiculi graciles and cuneati. Some degeneration of pyramidal tracts and of Gower's tracts. Improvement of all parts after passing upper level of olives.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Vessels of cord healthy and distended with blood-cells, but no exudates. No perivascular whorls in glial tissue. Abnormalities, probably gliomatous, present in dorsal and cervical regions of cord.

SUMMARY OF AUTOPSIES ON DOUBTFUL CASES.

Case 1.

A.—*Duration.*—Strasik Josepha; duration 7 years. Death at 23. Mitral disease, and probably tuberculosis present.

B.—*Author.*—Kahler and Pick. *Archiv für Psychiatrie*, Bd. viii., s. 251.

C.—*Cord.*—Small.

D.—*Memb.*—Adhesion of dura and pia.

E.—*P. Col.*—Degenerated throughout, except a thin zone next grey matter.

F.—*L. Col.*—Posterior parts degenerated up to periphery. Lateral limiting layer unattacked. Crossed pyramidal tract more affected on right side than on left. Direct cerebellar tract degenerated.

G.—*A. Col.*—Right side degenerated down to dorsal region; left unaffected. (The reporters suppose there was an anomalous arrangement of decussating fibres).

H.—*P. Horn.* ⎫ No change in grey matter beyond a relatively feeble
J.—*A. Horn.* ⎭ development.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Anterior normal. Posterior sclerosed.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*—Normal.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*

Case 2.

A.—*Duration.*—Marie R.; duration 8 years. Died at 32 of phthisis.

B.—*Author.*—Brousse. *De l'ataxie héréditaire*, Paris, 1882.

C.—*Cord.*—No mention of size.

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Most marked in lumbar region, and least in dorsal.

F.—*L. Col.*—Posterior parts sclerosed but not up to the periphery. Most marked in cervical and lumbar regions.

G.—*A. Col.*—Turck's column unaffected, but sclerosis present on both sides around heads of anterior cornua. Most extensive on left side.

H.—*P. Horn.*

J.—*A. Horn.*—Tips of cornua slightly affected in cervical region.

K.—*Clarke's Col.*—Not mentioned.

L.—*Sp. Roots.*—Not mentioned.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*—"Inflamed." Lumen diminished by a collection of small embryonal cells. Complete obstruction in lumbar region.

P.—*Med.*—Slight sclerosis in posterior pyramids.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Apoplectiform attacks present in this case.

Case 3.

A.—*Duration.*—N. N., female; duration 4½ years. Died at 29 of pulmonary phthisis.

B.—*Author.*—Auscher. *Archives de Neurologie*, 1890, p. 475, and *Semaine Medicale*, July, 1890.

C.—*Cord.*—Small.

D.—*Memb.*—Normal.

E.—*P. Col.*—Sclerosed. (A neuroglial sclerosis with integrity of the vessels and pial prolongations.) Lissauer's zone intact.

F.—*L. Col.*—Unaffected.

G.—*A. Col.*—Unaffected.

H.— *P. Horn.*—Atrophied.

J.—*A. Horn.*

K.—*Clarke's Col.*—Atrophy of fibres and diminution of cells.

L.—*Sp. Roots.*—Posterior roots contain many "embryonal" fibres, but no empty sheaths.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Sensory nerves show no degeneration of tubes or empty sheaths, but a great number of fibres with embryonal characters.

O.—*C. Canal.*—Replaced by a mass of epithelial cells.

P.—*Med.*

Q.—*Cbl.*—Small, but not degenerated.

R.—*Cbr.*—Small.

S.—*Remarks.*

Case 4.

A.—*Duration.*—Eg. Pall.; duration 6 years. Death at 18 from chronic pneumonia followed by marasmus.

B.—*Author.*—Tedeschi. *Beiträge zur Pathologischen Anat. und Allgemein. Pathol.*, Bd. xx., Hft. 1, 1896.

C.—*Cord.*—Small.

D.—*Memb.*—Dura normal. Pia thickened in posterior part.

E.—*P. Col.*—Sclerosed. Degeneration extending out to middle root zone. Escape of cornu commissural zone, and to a less extent of postero-internal root zone. Lissauer's tract attacked.

F.—*L. Col.*—Degenerated fibres in cervical region in area of D. C. T. At other levels merely abnormal thinness of nerve fibres.

G.—*A. Col.*—Abnormal thinness of nerve fibres.

H.—*P. Horn.*
J.—*A. Horn.* } Thinning of nervous reticulum. Diminution and pigmentation of cells. Those of posterior horn shrunken and deformed.

K.—*Clarke's Col.*—Degeneration of cells. Thinning of fibrillar plexus.

L.—*Sp. Roots.*—Atrophied. The various fasiculi composed of extremely fine fibres which react very feebly with specific stains. Interstitial tissue increased.

M.—*Sp. Ganglia.*—Striking thinness of nerve fibres. Axis cylinders and myelin sheaths stain badly with their proper stains. Some cells vacuolated, some over-pigmented. Interstitial tissue increased.

N.—*Peri. Nerves.*—Not examined.

O.—*C. Canal.*—Double in lumbar region, becoming single at higher level.

P.—*Med.*—Sclerosis of Goll's and Burdach's funiculi. Longitudinal fibres diminished. Horizontal ones diminished and thinned. Cells less numerous, shrunken, and pigmented. Reil's band appears unaltered both at origin and crossing. Pyramids, arciform fibres, and crura cerebri normal.

Q.—*Cbl.*—Normal size. Increase of pigment in Purkinje's cells. Thinning of fine plexus in nuclear layer.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The patient was insane.

LITERATURE.

Autopsies on Friedreich's disease, including doubtful cases of the same.

[1] Friedreich, *Virchow's Archiv*, Bd. xxvi., s. 329; *ib.*, Bd. xxvii., s. 1; *ib.*, Bd. lxx., s. 140.
[2] Schultze, *Virchow's Archiv*, Bd. lxxix, s. 132.
[3] Kahler and Pick, *Archiv für Psychiatrie*, Bd. viii., s. 251.
[4] Brousse, *De l'ataxie héréditaire*, Paris, 1882.
[5] Everett Smith, *Boston Medical and Surgical Journal*, 1885.
[6] Newton Pitt, *Guy's Hospital Reports*, 1887, p. 369.
[7] Rutimeyer, *Virchow's Archiv*, Bd. cx., s. 215.
[8] Dejerine and Letulle, *La Médecine Moderne*, April, 1890.
[9] Blocq and Marinesco, *Archives de Neurologie*, May, 1890.
[10] Auscher, *La Semaine Médicale*, July, 1890, and *Archives de Neurologie*, 1890, p. 475.
[11] Guizetti, *Riforma Medica*, June, 1893, and *Il Policlinico*, 1894, pp. 438 and 453.
[12] Mirto, *Giorn. del' Assoc. dei Medici e Natural.* anno iv., 1893.
[13] Burr, *University Medical Magazine*, Philadelphia, June, 1894.
[14] Dana, *Postgraduate*, New York, July, 1896.
[15] Tedeschi, *Beiträge zur Path. Anat. und Allgem. Path.*, Jena, Hft. 1, 1896.
[16] Simon, *Le Progrès Médical*, September, 1897.
[17] Bonnus, *Nouvelle Iconographie de la Salpêtrière*, No. 3, 1898.

Autopsies on cases of the cerebellar type:—

[18] Nonne, *Archiv für Psychiatrie*, Bd. xxii., s. 273. (Case of the brothers Stüben. Cerebella small, but not atrophied. Purkinje's cells normal.)
[19] Menzel, *Archiv für Psychiatrie*, Bd. xxii., s. 160. (Case of cerebellar atrophy, with cord changes similar to those of Friedreich's disease).
[20] Fraser, *Glasgow Medical Journal*, 1880. (Cerebellar atrophy, with normal condition of cord.)
[21] Meyer, Brain, part lxxix., vol. xx. (Autopsy on one of Sanger Brown's series. No circumscribed lesion in cerebellum. Degeneration of posterior columns, and of D. C. T. in cervical cord.)

The following have also been referred to:—

[22] Wollenberg, *Archiv für Psychiatrie*, Bd. xxiv., s. 213. (Changes in spinal ganglia in tabes. With plates and literature.)

[23] Lenhossék, *Archiv für Psychiatrie*, Bd. xxix., s. 345. (Normal histology of spinal ganglion cells. With plates and literature.)

[24] Gudden, *Archiv für Psychiatrie*, Bd. xxviii., s. 643. (Changes in peripheral nerves in neuritis. With summary of literature on segmental neuritis and regeneration processes.)

[25] Westphal, *Archiv für Psychiatrie*, Bd. viii., xiv., and xvi.

[26] Sakaky, *Archiv für Psychiatrie*, Bd. xv., s. 584.

[27] Nonne, *Archiv für Psychiatrie*, Bd. xix., s. 352.

[28] Oppenheim and Siemerling, *Archiv. für Psychiatrie*, Bd. xviii., s. 98.

[29] Dejerine, *Archives de Physiol. norm. et path.*, 1884.

[30] Dejerine, *Archives de Physiol. norm. et path.*, 1887. Also numerous papers in the same archives, vols. ii.-x., by Hayem and Gilbert, Babinski, Pierret, Pitres and Vaillard, Joffroy, Vignal and Gombault.

[31] Fleming, Brain, part lxxvii., vol. xx.

[32] Weigert, *Centralblatt für Allegemein. Path.*, Nov., 1890.

[33] Weigert, *Beiträge zur Kentniss der normalen menschlichen Neuroglia*, Frankfurt, 1895.

[34] Mallory, *Centralblatt für Allgemein. Path.*, Oct., 1895.

[35] Mallory and Wright, *Pathological Technique*, Philadelphia, 1898.

[36] Marie, "Lectures on Diseases of Spinal Cord," Syd. Society's translation, 1895.

[37] Gombault, *Comptes rendus de l'académie des sciences*, 1886, p. 439.

[38] S. Mayer, *Zeitschrift für Heilkunde*, bd. ii., 1886.

[39] Pitres and Vaillard, *Archives de Neurologie*, vol. xi., 1886.

[40] Erlicki and Rybalkin, *Archiv für Psychiatrie*, Bd. xvii., p. 709.

[41] Mitchell Clarke, *Brit. Med. Journ.*, Dec. 8, 1894.

[42] Mackie Whyte, Brain, part lxxxi., 1898.

[43] Ferrier, "Functions of Brain," 1886.

DESCRIPTION OF PLATES.

(*Staining in each case by Schäfer's method. Figs.* 1-5 *from microphotograph and sketch. Figs.* 6 *to* 8 *microphotograph only. Figs.* 1-5, × 10 *diameters. Fig.* 6, × 8 *diameters. Figs.* 7 *and* 8, × 70).

Fig. 1.—T. S. at L. 4. Sclerosis of posterior column, except in anterior fourth. Also of posterior roots, root-zones, and cornua. Escape of fibres in position of Flechsig's *centrum ovale*. Slight degeneration of C. P. T. on each side.

Fig. 2.—T. S. at L. 1. Sclerosis of posterior columns, with exception of fibres in position of cornu-commissural tract. Also of posterior roots, root-zones, and cornua. Sclerosis of Clarke's Columns. Degeneration of C. P. T.

Fig. 3.—T. S. at D. 6. Structural abnormality in posterior column (developmental ?).

(*a*) Semilunar area of sclerotic tissue, surrounded by a whorl of degenerate myelinised fibres.

(*b*) Capsule of sclerotic tissue enclosing a central medullated (degenerated) area, and in its turn enclosed by a layer of degenerated medullated fibres.

These latter probably represent those previously occupying the ventral fields of the posterior column, and bounding the posterior cornua.

FIG. 4.—T. S. at D. 2. Sclerosis of posterior column here reaches its maximum. Commencing improvement in root fibres, and in postero-external fields of Burdach's column. Atrophy of posterior cornua and Clarke's columns. Sclerosis of C. P. T., D. C. T. and D. P. T.

FIG. 5.—T. S. at C. 5. Abnormality in left posterior cornu. Structure somewhat similar to that described in fig. 3. Sclerosis of Goll's column. Improvement in postero-external fields of Burdach's columns. Well-stained fibres adjoining posterior cornua. Sclerosis of C. P. T., D. C. T. and D. P. T.

FIG. 6.—T. S. Medulla at decussation of fillet layers.

(*a*) Funiculus gracilis, and nucleus, sclerosed.

(*b*) Funiculus cuneatus, partially sclerosed, with poorly developed nucleus.

(*c*) Substantia gelatinosa. The fibrillar reticulum of the posterior cornu is fairly well myelinised.

(*d*) Direct cerebellar fibres, sclerosed.

(*e*) Gower's tract, slightly attacked.

(*f*) Fillet layers. Well-stained and healthy.

(*g*) Decussation of internal arcuate fibres. Their further course from posterior horn and Burdach's nucleus does not appear well in this section.

(*h*) Asymmetry and partial degeneration of anterior pyramids.

FIG. 7.—T. S. Sciatic. Schäfer. × 70.

FIG. 8.—T. S. Median. Schäfer. × 70.

A CASE OF TUMOUR OF THE PONS.

BY J. DIXON MANN, M.D., F.R.C.P.
Physician to the Salford Royal Hospital,

AND

SHERIDAN DELÉPINE, M.B., B.Sc.
Professor of Pathology in The Owens College.

WILLIAM S., aged 52 years, was admitted into the Salford Royal Hospital on August 11, 1893. He enjoyed good health until March, 1892, when he began to have shooting pains in the back of the head; the pains were subsequently felt at the top and the front of the head. He vomited occasionally. In December, 1892, he began to have humming noises in the left ear. In April, 1893, he felt a pain in the right shoulder, which extended down the arm to the hand. He has muscular twitches in the left leg and he feels rather dizzy. After remaining in hospital for several weeks, he was discharged at his own request, but he continued to attend as an out-patient. The right arm and right leg now began to feel weak, and in September, 1894, he had ptosis of the left eye, which lasted three months. In the early part of December he had a succession of convulsive attacks. The noise in the left ear has been continuous.

On February 4, 1895, he was re-admitted, when his condition was as follows:—The right arm, flexed at a right angle, was spastically contracted and devoid of motor power. The right leg was also spastic and devoid of motor power. There was no paralysis of the facial muscles and the ptosis had disappeared —the left eye could be freely opened. There was no perceptible paralysis of the ocular muscles—the eye ball was capable of the usual movements, nor was there any nystagmus. The pupils were equal, not contracted, and they reacted to light. There was double optic neuritis and slight limitation of the field of vision on the temporal side of the left eye; vision was otherwise unaffected. The humming or buzzing sound in the left ear was now associated with impaired audition on the same side. The perception of acid substances applied to the tongue was equally good on both sides. The pain in the head had almost disappeared. When tested with the point of a pin and with test-tubes respectively filled with hot and cold water, sensation was found to be unaffected on both sides of the face, on the arms and on the legs. On the right side the knee-jerk was slightly increased. The speech was much impaired, being thick and "bulbar"; there was also some difficulty in swallowing. The mental faculties were unaffected.

Whilst in hospital the patient had twenty to thirty seizures, in which, for the most part, the head and the eyes were turned to the right side, the left eye being less deflected than the right. When the patient was in a passive condition, no difference was perceptible between the movements of the left eye and those of the right. The seizures were characterised by a condition of tonic spasm, which lasted from a few seconds up to two or three minutes, the right arm and the right leg being absolutely stiff and rigid. The left arm and leg were not affected. The pupils were dilated and did not respond to light. The respirations were slowed. During the seizures and for a varying period afterwards—from a few minutes to half an hour—the patient was quite unconscious. On one or two occasions there were slight indications of an epileptiform character, and once, slow clonic spasms—one every four or five seconds—were observed to affect the right arm. In two of the seizures the right side of the face appeared to be partially paralysed, the cheek "flapping" with the respirations at the maximum of the attack. In February, 1895, both sides of the face, both arms, and both legs twitched during an attack. On another occasion the twitching was limited to the fingers of the right hand, the face not being affected. On March 21 the left eye was observed to remain closed for about twelve hours after an attack. During the interval between the attacks, if the patient (who was then quite conscious and intelligent) was asked a question, the answer to which involved some reflection, the left eyelids usually twitched convulsively. After a seizure on March 27, ptosis on the left side recurred and persisted until death, which took place on April 4, 1895. No sugar was found in the urine, nor albumin until a fortnight before death.

Professor Delépine's Report on the brain, cerebellum, pons and medulla oblongata.

These parts having been kept in weak spirit for a few days before being available for examination were very soft, and had partly lost their original colour. The only evidences of disease which could be detected by an external examination were the following:—

(1) A marked swelling of the left side of the pons, most distinct above the exit of the 5th nerve, and causing much asymmetry of the pons.

(2) A very considerable enlargement of the left peduncle of the brain.

(3) Great displacement of the basilar, left posterior cerebral and left superior cerebellar arteries as well as of the left 3rd and 4th nerves, and to a lesser degree of the 5th nerve.

(4) Atrophy of the left 3rd nerve, which measured about 2 mm. in diameter, whilst the right 3rd measured over 3 mm. in diameter.

(5) On separating slightly the left tempero sphenoidal lobe from the left crus, a tumour became apparent. Owing to the soft state of the lateral portion of the left crus, and of consequent lacerations of this part, the superficial relations of this tumour could not be very clearly ascertained. It was, however, evident that the tumour, which seemed to be of the size of a pigeon's egg, must have compressed the posterior parts of the left optic tract.

(6) When the upper surface of the cerebellum was exposed, a distinct bulging was evident in the anterior part of the left quadrate lobe. This swelling was situated about ½ inch to the left of the superior vermiform process, which was displaced towards the right, so that, seen from above, the two halves of the cerebellum seemed unequal. The left cerebellar hemisphere looked larger than the right.

Owing to the soft state of the specimen, it was not found possible to make many sections through the parts affected. The following were, however, found sufficient to determine with tolerable accuracy the localisation of the

Diagram I.

Diagram of left aspect of medulla, pons, crus cerebri, thalamus opticus, etc., to show the relations of the tumour and surrounding areas of degeneration as well as the direction of the various sections described in the text.

1 to 6.—Planes of sections. *II. to VIII.—Cranial nerves.*

A—Pulvinar.
B—External and internal geniculate bodies.
C—Pineal gland.
D—Corpora quadrigemina.
E—Peduncle of cerebrum.
F—Left corpus albicans.
G—Superior peduncle of cerebellum.
H—Middle ,, ,, ,,
K—Inferior ,, ,, ,,
L—Medullary centre of left hemisphere of cerebellum. The letter is placed at the confluence of the primary medullary lamellæ of the anterior part of the antero-superior lobule and of the lateral part of the central lobule.
M—Olive.

The tumour is indicated in black, and the degeneration by light shading.

Diagram II.

Section 1 (seen from behind).

A—Internal capsule.
B—Substantia nigra.
C—Optic thalamus.
*C**—Left optic thalamus at the place where degeneration has extended from the tegmental portion of the peduncle (degenerated part indicated by the darker shade).
D—Lenticular nucleus.
E—Velum interpositum.
F—Fornix.
G—Corpus callosum.

This sketch should be reversed to make it comparable with the other drawings.

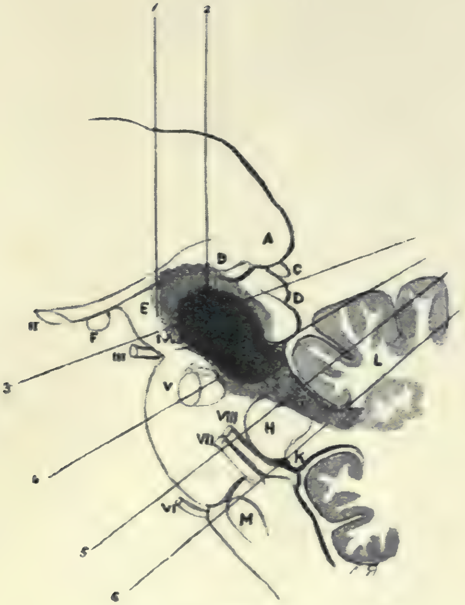

Diagram I.

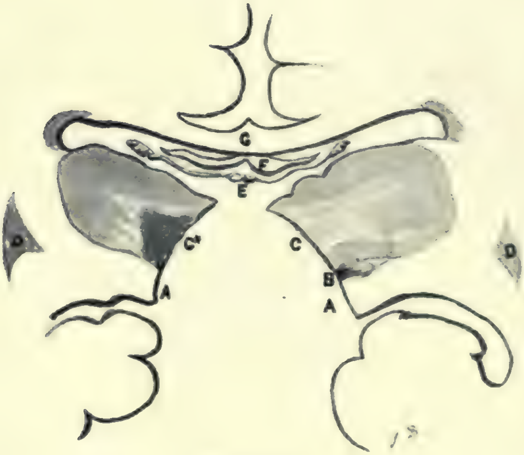

Diagram II.

lesions, except in the left external part of the left crus, which had been destroyed by softening and *post-mortem* laceration.

The planes of section will be best understood by reference to the diagram I., which represents the posterior parts of the basal ganglia, crura cerebri, pons varolii, medulla, cerebellum, and peduncle of the cerebellum, seen from the left side. In this diagram, the affected parts are shaded, the tumour itself deeply, the softened parts less deeply. All these parts could not be seen from the surface, as has already been explained.

Section 1 (Diagram II.).—A vertical frontal section passing through the hemispheres of the brain and dividing the basal ganglia behind the grey commissure. It shows the relations of the tegmental and crustal portions of the cerebral peduncles to the region of the optic thalami and the internal capsula. A small portion of the locus niger indicates the line of demarcation between the tegmentum and crusta. A patch of degeneration is seen to have destroyed a small portion of the left optic thalamus and subthalamic region. The internal capsule is apparently unaffected.

Section 3 (Diagram III.).—This section was slightly oblique, dividing the crura just above the superior corpora quadrigemina, and the point of exit of the 3rd nerves.

A very vascular tumour, surrounded by a layer of very soft, degenerated, nervous tissue, occupies the space between the grey matter of the corpora quadrigemina posteriorly, and the crusta anteriorly, the mesian plane on the right, and the surface of the peduncle on the left.

It is at this level that part of the tumour and surrounding tissues had been accidentally destroyed, but the shape and relations of the tumour indicate that it had probably extended from the surface of the crus, and originated in the meningeal covering. There can be little doubt either that the left corpora geniculata were involved. The left crusta, except in its most external part (temporo-occipital cerebellar fibres and pyramidal fibres for the leg), was not involved, but must have been subjected at times to considerable pressure. The grey matter round the aqueduct had been simply dis-

DIAGRAM III.

Section 3.—Passing through the superior corpora quadrigemina, the aqueduct, the crusta, a little above point of exit of the third nerves (seen from above).

A—Superior corpora quadrigemina.
B—Brachium of left testis.
C—Crusta; letter placed opposite pyramidal fibres.
D—Substantia nigra.
E—Aqueduct.
F—Right third nerve.
F'—Left third nerve.
The relative size of the two nerves is represented by means of transverse sections, below the main diagram.
G—Basilar artery with its branches.
H—Tumour (dark shade).
I—Zone of softening round the tumour (lighter shade).

DIAGRAM IV.

Section 4.—Passing through pons Varolii at level of exit of fifth nerve, upper part of superior cerebellar peduncles just below the inferior corpora quadrigemina, inferior corpora quadrigemina, and most anterior and superior cerebellar laminæ (seen from above).

A—Superior cerebellar peduncle.
B—Fifth nerve.
C—Pyramidal bundles.
D—Superficial transverse fibres of pons.
E—Deep transverse fibres of pons.
F—Fillet.
G—Aqueduct near upper end of fourth ventricle.
H—Anterior end of superior vermiform process.

Tumour and degenerated parts indicated as in previous diagrams.

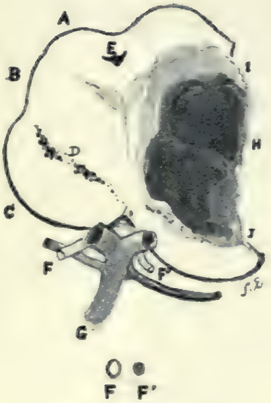

Diagram III.

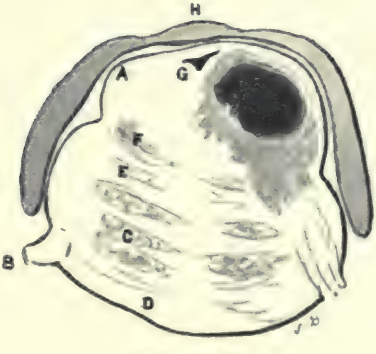

Diagram IV.

placed towards the right. It seems impossible that the fibres of the left 3rd nerve should have escaped destruction in their passage from their nucleus to the surface, and as a matter of fact the left 3rd was in a state of advanced atrophy.

Section 4 (Diagram IV.).—This section is slightly more oblique than the last, and followed the fibres of the 5th nerve for some distance through the pons.

In this section the lower part of the tumour is seen to extend beneath the fibres of the superior cerebellar peduncle, which seem to be partly destroyed by it. The zone of softening round the tumour extends more in the direction of the fibres of the 5th than in any other direction. The aqueduct is pushed towards the right, and part of the grey matter round it on the left side remains apparently unaffected. The right half of the tegmental and ventral parts of the pons are but slightly affected by pressure at this level, but the left half is still considerably enlarged.

Section 5 (Diagram V.).—This section is a little more oblique than the previous one. It is entirely below the level of the tumour, and shows only a tract of degenerated tissue in the region corresponding to the superior cerebellar peduncle and adjacent white matter. Though the left half of the pons is distinctly larger than the right, it seems almost entirely free from lesion.

Section 6 (Diagram VI.) is nearly parallel to the last. It passes through the upper part of the medulla oblongata, showing the exit of the 8th nerve; it also divides the floor of the 4th ventricle at about the level of the striæ acusticæ, it exposes the white centres of the cerebellum, with a small part of the corpora dentata; the relations of the central lobule are also well shown.

The only lesion visible at this level is a small patch of softening evidently following the direction of the fibres of the superior cerebellar peduncle and involving the region of the left corpus dentatum. There is a slight displacement of the central lobule towards the right; the floor of the 4th ventricle may have been at times compressed by the central lobule when the diseased parts underwent rapid enlargement from one cause or another.

Diagram V.

Section 5.—Passing through the pons Varolii at a level half way between lower edge of the pons and exit of the fifth nerves (seen from above).

A—Superior medullary velum, behind fourth ventricle.
B—Middle peduncle of cerebellum.
C—Seventh and eighth nerves.
D—Pyramidal fibres.
E—Flocculus.
F—Superior or quadrate lobe.
G—Vermiform process.

Softened part shaded as in previous diagrams.

Diagram VI.

Section 6.—Passing through medulla immediately below lower border of pons, through the central lobule and postero-superior lobule of cerebellum (seen from above).

A—Anterior pyramid.
B—Root of eighth nerve.
C—Central lobule.
D—Upper part of right corpus dentatum.

The patch of softening is indicated as in other diagrams.

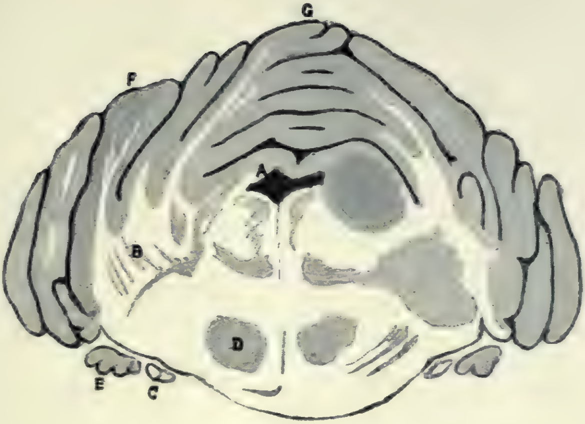

Diagram V.

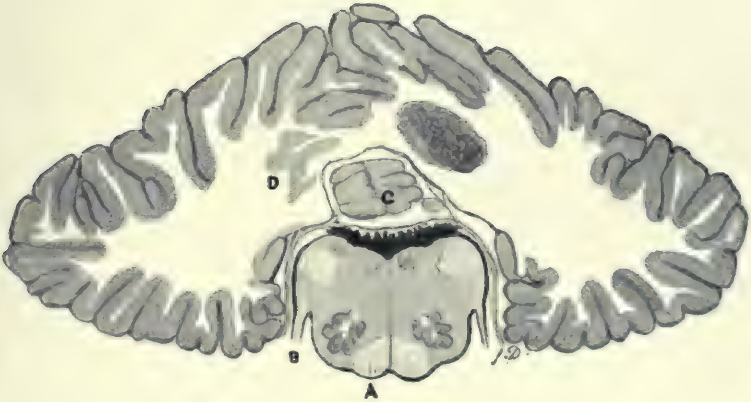

Diagram VI.

From a study of these sections one may safely come to the following conclusions :—

The left crus was the seat of a tumour which had destroyed the greater part of its tegmentum. This tumour had apparently originated near the external left aspect of the crus, probably extending from the membranes and inserting itself like a wedge between the left corpora quadrigemina and the crustal portion of the left cerebral peduncle, causing them to become widely separated and disconnected. This tumour did not extend upwards higher than the upper parts of the upper corpora quadrigemina, and downwards below the upper border of the middle cerebellar peduncle; the formatio reticularis and the red nucleus were the parts primarily invaded and destroyed by the tumour (within the region affected); other parts were also destroyed, *more or less* completely, being involved in the zone of degenerated and necrosed tissues surrounding the tumour; these were the left superior cerebellar peduncle, the left upper and lower fillet, the left corpora geniculata and posterior part of the left optic tract, the fibres of the root of the left third nerve; the left 4th nerve was possibly involved, in its course round the diseased crus. Many of the fibres of the left 5th must also have been involved.

The nuclei of the 3rd, 4th, 5th, were all in close contact with the tumour, and partly involved in the necrosed zone.

The fibres of the 2nd, 3rd, 4th, 5th, 6th, 7th, 8th nerves had either to pass through degenerated zones or were subjected to pressure, which must have been very variable in degree, considering the very vascular nature of the tumour. The external ¼th of the left crusta was partly infiltrated by the tumour, and had certainly much suffered from pressure; the rest of the left crusta was also subjected to pressure, though in a lesser degree. The pressure must also have at times affected indirectly the right half of the pons and the right crus.

The left 3rd nerve was the only nerve which showed striking evidences of the destructive action of the tumour.

This nerve was much atrophied; its diameter was only ⅔rds of that of the corresponding nerve on the right side.

On microscopical examination not more than $\frac{1}{4}$th of its fibres could be recognised, and most of these were in a state of degeneration. Connective tissue occupied the space originally occupied by the other fibres. The right third was also partly degenerated, not more than $\frac{2}{3}$rds or $\frac{3}{4}$ths of its fibres presenting a normal appearance.

The tumour, by its situation, its relations to the formatio reticularis, the apparent hypertrophy of the pons produced by it, might have been supposed to be a typical glioma; but it was quite clearly a round-celled sarcoma. It was well defined, surrounded by a well marked zone of necrosed tissue, so soft that it was difficult to prevent a displacement of the parts. The tumour was nodulated, the nodules being firmly connected together by the condensed tissue intervening between them. The nodules were soft, very vascular, and owing to their dark colour, contrasting sharply with the surrounding opaque, yellowish white, surrounding necrosed nerve tissue. There was enough of this softened tissue to reconstitute the parts of the crus which seemed to have been replaced by the tumour, so that it is very doubtful whether there was any organic connection between the new growth and any part of the tegmentum. The cells of the tumour were small, rounded, with large nuclei, not separated by any distinct stroma except quite at the periphery of the nodules, where, through pressure, the cells were arranged in rows or packets separated by a very small amount of a clear, slightly fibrillated matrix.

The vessels were large, most of them with indistinct walls composed of embryonic looking cells; in some places the vessels were surrounded by several concentric layers of these cells. Though the tumour was almost angiomatous in a few places, there were but few traces of hæmorrhage; there was little evidence of degeneration in the central parts of the nodules. In all these respects the tumour resembled closely similar sarcomata growing from the soft membranes of the brain.

I therefore come to the conclusion that the case is one of round-celled sarcoma (very vascular) growing from the pia mater and penetrating into the left peduncle of the brain, causing destruction of the tegmentum at that level, pressure on surrounding parts, and degeneration of certain fibres.

On reading Professor Delépine's description it is a matter for surprise that so many of the cranial nerves escaped suffering from the effects of the lesion; for not only were most of them directly involved by the tumour, but many had also to pass through a zone of degeneration and were subjected to varying pressure due to great vascular irregularity of the degenerated area. The third was the only nerve which showed definite atrophy, the paralytic symptoms being very partial; ptosis occurred, but no external strabismus, nor any alteration in the size or mobility of the pupil, nor in the power of accommodation. Lesions of the third nerve, however, often produce only partial results. No facial pain nor anæsthesia occurred from first to last, although the fifth nerve and its nucleus came well within the influence of the tumour; the seventh nerve also escaped, notwithstanding its dangerous contiguity. The slight indications of paralysis of the right side of the face on two occasions were due to diffuse pressure from increased vascularity. On the other hand, subjective auditory sensations were among the first and the most persistent of the symptoms; this, in a degree, is common, as are also spasm and convulsions in chronic lesions of the pons, the very gradual compression of the fibres of the pyramidal tract rarely causing sufficient irritation as to evoke spasm. The extremely slow growth of the tumour—three years and one month from the earliest symptom—probably accounts for the extraordinary tolerance displayed to its disturbing influence by the cranial nerves.

A FORM OF DISEASE RESEMBLING THE PSEUDO-SCLEROSIS OF WESTPHAL AND STRÜMPELL.

BY WILLIAM G. SPILLER, M.D.

Professor of Diseases of the Nervous System in the Philadelphia Polyclinic; Associate in the William Pepper Clinical Laboratory, University of Pennsylvania.

From the William Pepper Clinical Laboratory.

So few cases of this peculiar disease, described by Westphal and Strümpell, have been reported, that the following case, observed at the Pennsylvania Training School for Feeble-Minded Children, seems to be worthy of some attention.

T. H. M., male, was born in March, 1882, and died May 21, 1897. His parents were Irish, and his father was a labourer. Both parents were in good health prior to the birth of this boy. The mother died after the birth of her third child; according to the report, from abscess. She was subject to violent headaches. The father had a feeble-minded brother and an insane sister.

T. H. M.'s elder brother died in convulsions during an attack of scarlet fever. T. was the second child in the family, and was born at full term in ordinary labour, and was nourished by the breast. His mother was 39 years old at the time of his birth.

Nothing peculiar was noticed in the boy until he went to school. At this period his mental condition was found to be below the average. He did not defend himself from the attacks of his comrades, and was tormented by other boys in the street. He was said to have a good memory for objects, as well as considerable imagination, and he was able to relate stories. His gait and speech were rather slow, and he had a slight lisp. His circulation was poor, and his hands, as a rule, cold.

At the time of admission to the Pennsylvania Training School for Feeble-Minded Children, March 16, 1893, he was of good size, with no striking peculiarities in the formation of the head. His sight and hearing were good. He was right-handed. No peculiar movements of face or body were noted. He did not laugh or weep without cause. He had no convulsions. He masticated his food well, was not gluttonous, and had fair table manners. He was quite active, not especially noisy, affectionate, and obeyed commands. He was trustful and truthful, tidy about his dress, and able to understand what was said to him.

April, 1893.—The boy could read quite readily at sight, and could count, but he had no knowledge of addition, and wrote a peculiar hand. He seemed interested in all the exercises the children were taught, though he at times appeared to be troubled because he could not always understand.

November, 1894.—After a long illness he re-entered school. He was retrograding, and seemed to have difficulty in going up and down stairs.

February, 1895.—Examination at this date showed impaired co-ordination, poor station, unequal pupils, and poor circulation. He had been gradually losing the use of his limbs.

December, 1896.—He had previously been able to sit in a chair, but could no longer do so, and fell forward when he attempted this. He had been ill several times. His legs were much atrophied, rigidity of all the joints was noted, and the hands and arms were so rigid and atrophied that he was unable to feed himself. His vocabulary had been gradually reduced to "yes" and "no," and later he was unable to utter these words.

April, 1897.—The muscular atrophy had become more marked. No improvement in his general condition had occurred. His limbs were contractured, the reflexes were much exaggerated, and the boy was unable to speak. Bed sores were present over both hips. He was unable to turn himself in bed.

Dr. Risley examined the boy's eyes, November 30, 1895, and found bilateral optic atrophy. It is not known how long this had existed.

Dr. White stated that the boy had a coarse tremor in voluntary movement. The impairment of gait was first noticed in the difficulty the boy experienced in going up and down stairs. His speech at first had been lisping, later became scanning, and finally was lost.

The preceding notes were furnished to me by Dr. Frank White from the case-books of the Pennsylvania Training

School for Feeble-Minded Children. I saw the boy only once; this was on May 11, 1897, at which time I made the following notes.

The reflexes all over the body were much exaggerated, distinct ankle clonus could not be obtained on account of the contractures, but a tap on the Achilles tendon of the right side threw the foot into a tremor, consisting of movements of flexion and extension. This was not obtained on the left side. A tap over either patellar tendon gave an exaggerated knee-jerk, although the leg was not moved to any great extent on account of contracture. The exaggeration was especially noticeable in the contraction of the thigh muscles. The reflexes in the upper limbs at the wrists and biceps tendons were exaggerated. The chin-jerk was also unusually prompt. The muscles of the extremities were much contractured, and the legs could be extended passively on the thighs only to a right angle. The right forearm could be extended passively so as to form an oblique angle with the arm. The left forearm could be fully extended passively. No voluntary movement could be detected. A fine tremor was produced by any passive movement of the right hand. Pain sense seemed to be preserved in all parts of the body, but cerebration was very slow, and no response in any form to questions could be obtained. The muscular wasting was extreme in all parts of the body, and the boy was scarcely more than skin and bones. He was entirely unable to stand, as his lower limbs were extremely flexed, and he had no power to extend them. The bowels were constipated and an enema was necessary to obtain any movement. The urine escaped involuntarily and constantly. He uttered no distinct words, but whined continually. Fibrillary tremor in the muscles when at rest was not observed, probably because the atrophy was too great.

The boy died ten days after this examination, and Dr. White was able to obtain permission to remove only the cord. As I was not on duty as pathologist to the institution at this time, I am indebted to my colleague, Dr. Joseph Sailer, for the pathological material.

Such is the clinical history of the case so far as it could be obtained. A boy, born at full term, in normal labour, in a family in which feeble mentality had been observed, presented nothing striking to his parents until he went to school. He had at first a fair amount of intelligence, and could read and write; had a lisping speech, and no very marked disturbance of gait. As he grew older mental failure, spasticity, tremor on movement, scanning speech, contracture of the muscles of the limbs, gradual abolition of the power of voluntary movement, and bilateral optic atrophy were noted. Most of these symptoms, according to the history, developed in the last three or four years of life.

The process was evidently one causing degeneration of the pyramidal tracts, and probably of the anterior horns of the spinal cord, on account of the intense muscular atrophy. It was a process of long duration—if we may assume that the boy went to school when he was seven or eight years old—lasting seven or eight years, inasmuch as the patient was fifteen years old at the time of death. Indeed, it is very probable that the degeneration began before the signs were noticed by the boy's parents and teachers.

It was, therefore, with considerable interest that I awaited the results of my microscopical examination. No one can regret more than the writer the inability to obtain the brain from this patient, but the spinal cord affords a very interesting study.

The crossed pyramidal tracts are degenerated from the medulla oblongata throughout the cord into the lumbar region, and some sclerosis is seen in the area of at least one direct pyramidal tract. The sclerotic tissue in the lateral columns extends forward somewhat beyond the area of the crossed pyramidal tracts, and is symmetrical on the two sides of the cord. These areas are not entirely degenerated, but contain many normal fibres. While this degeneration is unquestionably present, it does not equal in degree that resulting from a destruction of motor fibres at a higher level. The motor cells in the anterior horns of the cervical cord are nearly, or fully, as numerous as in normal cords, and most of them appear to be normal. The motor cells of the lumbar cord seem to be somewhat deficient in number. As the spinal cord was unfortunately hardened in Müller's fluid, Nissl's method could not be employed.

The anterior roots are certainly not notably altered. The anterior horns do not appear to be as rich in fibres as in normal spinal cords. The method of Marchi reveals some black dots in the sections, but not sufficient to render a diagnosis of recent degeneration probable.

These are, therefore, the lesions of amyotrophic lateral sclerosis, or more correctly, of lateral sclerosis, for the changes in the peripheral neurons are not marked, as they are in the central.

I cannot say that symmetrical gross lesions were not present in the motor regions of the brain, but the clinical history is much against such a view, and the degeneration of the pyramidal tracts equals in intensity that which I have found in amyotrophic lateral sclerosis, and is less, to my eye, than that known as secondary degeneration from cerebral lesions. This seems to me an important observation, although, of course, the age of the child must be taken into consideration. The boy was born in a family in which the mentality was feeble, and doubtless inherited a central nervous system which was not of the best. The gradual development of great spasticity and paralysis after he began to attend school, and the absence of these signs in a notable degree before this period, do not render the existence of bilateral hæmorrhage, softening or similar lesions probable. The clinical history and the pathological findings are much in favour of the diagnosis of amyotrophic lateral sclerosis, and yet this disease has been thought to be one of adult life.

Luce has reported a case which he says proves by histological investigation that tabes dorsalis and amyotrophic lateral sclerosis may occur in childhood,[1] and he further states that such a demonstration of the occurrence of amyotrophic lateral sclerosis in childhood has not been previously given.

The interesting cases reported by Werdnig[2] and Hoffmann,[3] though not typical clinically of amyotrophic lateral sclerosis, show that this disease may exist in childhood. Hoffmann, in his last paper on this subject reports a case which he says represents the disease very fully. I quote it, therefore, as representing Hoffmann's latest views on this subject.

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[1] Luce, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xii., No. 1.
[2] Werdnig, *Archiv. für Psychiatric*, xxii., *idem* xxvi.
[3] Hoffmann, *Deutsche Zeitschrift für Nervenheilkunde*, iii., *idem* x.

A child born of healthy parents, in normal labour, appeared to be in good health until seven or eight months old. Flaccid paralysis began at that time to be apparent in the muscles of the buttocks and thighs, and gradually extended symmetrically to the back, neck, shoulder, upper arm, forearm and hand muscles, as well as to the muscles of the legs. Atrophy and loss of tendon reflexes, and secondary changes in the joints and vertebral column were noted, but no fibrillary tremors were seen, and sensation and the sphincters were intact. The mentality was good. The facial, lingual and pharyngeal muscles were normal. Death occurred from secondary pulmonary disease. In other cases reaction of degeneration had been noticed.

The peripheral motor neurons below the hypoglossal nerves were symmetrically degenerated, the cells of the anterior horns were much diseased, and sclerosis of the crossed and direct pyramidal tracts, and of the lateral ground bundles was noted, but the degeneration did not extend above the pyramidal decussation.

Twenty-two cases of this peculiar hereditary form of muscular atrophy, according to Hoffmann, are on record, and necropsies have been obtained in four of these. In all four the peripheral motor neurons were much diseased; in three the antero-lateral columns were affected, though in less degree, and in all the cases examined *post-mortem*, simple muscular atrophy was noticed.

The heredity, so far as known, the flaccid paralysis and loss of tendon reflexes were not present in my case, and the explanation for the existence of the flaccid paralysis and loss of reflexes in the other cases seems to lie in the greater degeneration of the peripheral neurons.

The case of T. H. M. resembles more the disease which Strümpell[1] has recently made prominent. He reports two cases of this malady which began in childhood and resembled multiple sclerosis, but no very marked lesions were found at the necropsy. He refers to two similar cases reported by Westphal in 1883. These are the only instances known in literature. The disease has commenced at the thirtieth, eighteenth, twelfth, and ninth year of life. The cause is unknown, but does not seem to be hereditary or familial. The patients have usually been normal before the beginning of the malady. The motility of the limbs is especially affected (spasticity, paresis, ataxia, tremor), in this pseudo sclerosis. Complete and permanent paralysis is rare, or at least late in appearing, but transitory paresis is very characteristic, and in some cases apoplectiform attacks with temporary hemiplegia are observed. The tremor is prominent and occasionally is of a rhythmical character. Distinct nystagmus was not seen in any of the cases. Scanning, indistinct and explosive speech was observed. The facial muscles appeared in some cases peculiarly rigid. Epileptiform attacks have been observed. The muscular tonus is exaggerated especially in the legs, and the gait is paretic, ataxic, and spastic. Exaggeration of the tendon reflexes is characteristic. Sensation is little affected. Vertigo and disturbance of the vesical, rectal and sexual functions are not usually observed. Mental symptoms are never entirely absent, and forced laughter and crying are noted. The duration of the disease is long and remissions occur. It does not resemble hysteria.

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[1] Strümpell, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xii., No. 2.

The findings in Strümpell's two cases were negative, except that a very slight degeneration of the upper part of the lateral motor tracts of the cord was observed, but this was not sufficient to explain the symptoms. In two of the four cases the white matter of the brain was unusually firm.

Strümpell reports another case which he observed in 1879-1881, in a child of 11 years, when first seen, which resembles the pseudo-sclerosis. At the necropsy the consistency of the brain was greater than normal, and the crossed pyramidal tracts were slightly degenerated throughout the cord. It is possible that syphilis bears some relation to the disease.

Strümpell, therefore, observed slight degeneration of the lateral motor tracts of the cord. In my case this degeneration was very marked, but the difference histologically between Strümpell's cases and my case seems to be largely one of degree.

The clinical history in my case pointed to a symmetrical progressive degeneration of the motor tracts, and the necropsy did not disappoint the expectations. If any cortical lesion was present it has escaped detection, and the report is imperfect to that extent. Such clinical cases are rare, and I feel justified in reporting this one, although the histological study was of necessity deficient.

The optic atrophy and the progressive mental enfeeblement bring to mind the affection first described by Warren Tay, and now known widely as amaurotic family idiocy of Sachs. Some of the other features are unlike the published cases of this disease.

It might be allowable to class this case among the cerebral diplegias, and there can be no doubt that the central nervous system was abnormal. Encephalitis might be thought of, but the clinical history does not seem to harmonize with such a diagnosis, unless we speak of the chronic form of this disease, which is not always easily distinguishable from primary degeneration of nerve cells and nerve fibres.

ON PARALYSIS AND MUSCULAR ATROPHY IN TABES DORSALIS, WITH OBSERVATIONS ON THE RELATION OF AFFERENT IMPULSES TO MOVEMENT AND MUSCULAR NUTRITION.

BY ARTHUR J. WHITING, M.D., M.R.C.P.
Registrar to the National Hospital for the Paralysed and Epileptic, Queen Square.

ALTHOUGH most of the prominent symptoms of locomotor ataxia are sensory, or at least afferent in their nature, as for example the anæsthesia and the lightning pains, there are yet some quite characteristic effects of the disease that are motor, or at least efferent, as for example the muscular palsies and atrophy concerning which this paper is written, and even, in the last analysis, the ataxy. In 1875 Hughlings Jackson[46] suggested that the ataxia of tabes is due to paralysis of certain highly special movements; but in 1896,[47] returning to the same subject, he writes, "I should have said paralysis, or rather, paresis *in effect*, the negative central lesion being a sensory one. There is in the early stage of the effort to walk (which effort, the operation being started by 'volition,' is from the highest cerebral centres) underdoing of the *movement* in which the peroneus longus *muscle* is chiefly engaged, and there is overdoing of the antagonist movement, that in which the tibialis anticus is chiefly engaged. I suppose the motor centre (lowest level) for the peroneal movement is not, consequent on the abrogation of the muscular sense nerves, 'informed' of the state of the muscles concerned in the movement, and is thus not kept 'ready to act,' and does not act efficiently in locomotion at the call of the highest centres. There is 'paralysis in effect.' On strong efforts to make it act the associated centre for the tibialis anticus movement is made to overact."

According to Erb, Topinard, and Cyon,[2] distinct motor paresis can be demonstrated in at least one-half of all cases of tabes, while Duchenne found muscular atrophy in 8 per cent. of his cases, and Dejerine[33] in 19 per cent. of his. It was in 1832 that Cruveilhier[1] first described atrophy of the lower limbs and double club-foot associated with sclerosis of the posterior columns of the cord. Since then cases have been recorded in which there has been atrophy involving, in different patients, almost every muscle of the body. A more or less general atrophy, usually resembling the Aran-Duchenne type of progressive muscular atrophy, has been observed in tabetic cases by Virchow, Foucart and Duchenne, Friedreich, Charcot, Buzzard, Ballet, Eulenburg, Hacker, Fischer, Dejerine and Braun; atrophy of the upper limbs, and usually of the hands by Cuffer, Ballet, Condoleon, Remak, Hoffmann, and Dejerine; of the lower limbs, and chiefly of those below the knee, by Laborde, Dumenil, Leyden, Joffroy and Condoleon, Stransky, Goldscheider, and J. B. Charcot with Dufour; of the tongue by Charcot and Cuffer, by Ballet, Westphal, and Eisenlohr; of one of the laryngeal muscles by Dufour; of the facial muscles by Ballet; and of the muscles of mastication by Schultze, of Bonn. Cases of paralysis of muscles inaccessible to direct physical examination during life, such as those of the eye and larynx and the diaphragm, have been recorded by a still larger number of observers. In the majority of the cases with autopsies the atrophy has been associated with a neuritis almost entirely terminal; in one case recorded by Remak[24] there was neuritis of the right median nerve, in one by Goldscheider[36] neuritis of the sensory and motor nerves of the lower limbs, in another by Dejerine and Petreen, in which there was external ophthalmoplegia and paralysis of the larynx, there was a parenchymatous neuritis not involving the nerves between their nuclei and the ponto-bulbar surface. Dejerine[33] has recorded nineteen cases of muscular atrophy occurring among ninety-three tabetics; in nine of these an autopsy was obtained, eight showing the Aran-Duchenne type of atrophy exhibited a well-marked neuritis principally of the intramuscular nerves of the extremities; the nerve changes were much less in the nerve trunks, and if present were only very slight in the anterior roots. In the ninth case, which was of the scapulo-humeral type, in addition to a neuritis there was, he thinks, a slight diminution in the number of anterior horn-cells in the cervical enlargement. In a minority of the cases with autopsies, the atrophy has been found to be associated with nuclear changes. Charcot[7] and Pierret[8] first directed attention to muscular atrophy in tabes accompanied with degeneration of the anterior horn-cells. There was in their well-known case atrophy of the muscles of the right upper limb, and more particularly those of the thenar and hypothenar groups, also of the muscles of the right side of the trunk and of the right lower limb. In this case the nerves themselves were healthy, and while the cells of the left anterior horn were quite normal, those of the right side, and especially the postero-external group, were greatly diminished in number, and those existing showed marked degenerative changes. They were able to trace degenerated fibres from the posterior roots to the anterior horns along the course of the so-called internal root-fibres, and they maintained that the cornual lesion had none of the characters of a polio-myelitis, but was in their opinion a degeneration secondary to that of the posterior columns.

In 1875 Cuffer[10] recorded a case of tabes in which during the prae-ataxic stage of the disease there had occurred atrophy of the right thenar muscles and right half of the tongue, with fibrillary twitching in the wasted tongue-muscles. An autopsy was made on this case in 1884 by Raymond and Artaud,[11] and it was found that in the dorsal and cervical regions of the cord the sclerosis had extended into the anterior horns, the cells of which were diminished in number, and those present shrunken, while in the bulb the right principal hypoglossal nucleus consisted of three or four cells alone as compared with thirty on the left side, and these three or four showed degenerative changes.

In 1876 Jean[9] recorded the case of a tabetic patient in which there were laryngeal and gastric crises, with spasm of the pharyngeal muscles and muscular twitchings of the lower limbs coinciding with severe lightning pains. There was found *post-mortem* atrophy of the left vocal cord and of the left thyro-arytenoid muscle, with degeneration of the left vagal and spinal accessory nuclei, and atrophy of their roots.

In 1877 a case of tabes, with muscular atrophy associated with anterior horn changes, was published by Leyden.[45] The atrophy involved the limbs, but more especially the hands, and the serratus magnus, the greater pectoral and the dorsal scapular muscles on each side. There was also observed fibrillary twitching in the thenar and tongue muscles. The anterior roots proved to be healthy; the anterior horn-cells of the cervical and lumbar regions were atrophied, and there was an intra-muscular neuritis.

In 1882 Buzzard[13] recorded a case of ophthalmoplegia externa occurring in a tabetic patient, and Bevan Lewis found a lesion of the sixth nucleus, which he believed to be thrombotic. In 1881 Kahler[12] found degeneration of the inferior vagal nucleus in a case of tabes, with paralysis of the muscles of the pharynx and right vocal cord, and with gastric crises. Landouzy and Dejerine[15] (1883) discovered atrophy of the vagus-accessorius nuclei in a case of tabes with intense laryngeal crises.

Eisenlohr[16] (1884) has described atrophy of the hypoglossal nuclei, the vagus-accessorius nuclei and the right sixth nucleus associated in a case of tabes with fibrillary twitchings and atrophy of the tongue, paralysis of the palate, lips, laryngeal muscles, and right external rectus muscle.

P. D. Koch, of Copenhagen, and Pierre Marie,[18] examined in 1888 the medulla from one of three cases of hemiatrophy of the tongue in tabes that had been recorded by Ballet[17] in 1881. In addition, there was in this case atrophy of the deltoids, of the muscles of the limbs (particularly the intrinsic muscles of the hands and those below the right knee), likewise paralysis of the soft palate and of the vocal cord on the same side as the wasting of the tongue. There was found atrophy of the right hypoglossal and spinal accessory nuclei. Westphal[27] published in 1887 a case of locomotor ataxia in which there was external ophthalmoplegia and atrophy of the left half of the tongue with weakness of the palate. Microscopic examination revealed degeneration of the third, fourth and sixth nuclei on each side, and of the left hypoglossal nucleus. In the same year Condoléon[26] made an elaborate study of a case of tabes with amyotrophy which had been previously recorded by Joffroy.[27] There was double club-foot; the muscles below both knees were wasted, particularly the peronei and solei, and more on the right side than the left; and there was atrophy of the intrinsic muscles of the left hand, more especially of the thenar and first interosseous muscles. In this case also severe lightning pains were often accompanied by involuntary contractions of the muscles, mostly of the hamstring group. Fibrillary twitchings were not observed. The electrical excitability of the wasted muscles was greatly reduced, but there was no characteristic reaction of degeneration. Degenerative changes were found in the anterior horn-cells of the eighth cervical and first dorsal segments of the left side, and their number was reduced, as compared with the right side, in the ratio of seven to ten. In the lumbar enlargement the anterior horn-cells were likewise decreased in number and degenerated, more, it was noted, on the right side than the left, corresponding with the muscular atrophy. The peripheral nerves were greatly degenerated, but the larger nerve-trunks and the anterior roots showed but slight changes. The degenerated posterior root-fibres could be traced as far as the anterior horns but not actually into their grey matter. A case in many respects similar was published by Braun[28] in 1888; there was in it atrophy of the intrinsic muscles of the right hand, the extensors of the right wrist and fingers, of both greater pectorals, the supra-spinati, infra-spinati and deltoids. The wasted muscles showed the reaction of degeneration, and fibrillary contractions were seen in those of the forearm. There was found *post-mortem* numerical diminution and degenerative changes in the existing anterior horn-cells of the lower cervical region with degeneration of the lower cervical roots on the right side, also of the right radial, left sciatic, and right peroneal nerves.

In 1896, Schaffer[44] examined with the aid of more recent methods the spinal cord from a case of tabes with club-foot and atrophy of the peroneal muscles. While the anterior horn of the cervical cord was normal in the lumbar region, Nissl's stain revealed chromatolytic changes in the cells, a finely granular chromatin with vacuolation, giving an appearance, he states, similar to that produced in the nerve cells by poisoning with phosphorus or lead. The anterior nerve-roots were found to be perfectly healthy.

The two following cases illustrating the occurrence of muscular atrophy in locomotor ataxia are those of two of Dr. Ferrier's out-patients, who kindly allows me to record them in this paper.

Case I.—B. C., aged 44, began to suffer from "rheumatic pains" in the lower limbs some twelve years ago. The pains, which were "momentary," "shooting," or "shock-like" in character, recurred usually every three or four months, each attack lasting for three or four days at a time. Four years later (1890) he began to have difficulty in getting his left hand to the back of his neck, experienced chiefly in his daily ablutions, and about the same time he noticed difficulty in lifting things from the table. Five years ago (1893) he first came as an out-patient to the National Hospital, when Dr. Ferrier found that he had greatly contracted pupils which did not react to light, absent knee-jerks, and paralysis with wasting of the flexors of the left forearm. It was also found that the biceps and supinator longus did not react to faradism, although they gave KCC>ACC, while all the other muscles reacted normally. His disability slowly increased, and five months later (December 6, 1893) the left deltoid and infra-spinatus were found to have lost their faradic excitability and to give ACC>KCC. On May 16, 1894, the biceps still gave KCC>ACC without any response to faradism.

About this time he began to have girdle sensation, as of a rope round his waist, and to have difficulty in passing water. On February 18, 1896, the infra-spinatus, deltoid, biceps, brachialis anticus, and supinator longus, of the left side, gave no reaction to either faradism or galvanism, but the left supra-spinatus reacted well to faradism and gave KCC>ACC, and KCC with 5 m.a. of current. Latterly the shooting pains have been more severe and have involved his arms as well as his legs; but he has throughout been free from all crises and has not had any diplopia or staggering in walking even in the dark.

There is nothing of importance in his family history. At the age of 20 he had a chancre with well-marked secondary manifestations. He has not had any other illness with the exception of influenza five years ago. He has always been a temperate man and his work has been carried on indoors.

He is a well-nourished, muscular man, about 5 feet 1 inch in height and 9 stone 6 lbs. in weight. Although his complexion is pale, there is no evidence of general anæmia. His pulse rate is 104 per minute ; it is small and slightly irregular, both in force and time. His rate of breathing is fifteen to the minute and is abdomino-costal in type. He is a quiet man, of good general conduct, without delusions or hallucinations, and is indeed of a considerable degree of intelligence. He can neither abduct nor advance the left humerus, nor flex the elbow joint, except very feebly by means of the long flexors of the wrist and fingers. When the elbow is extended the joint is locked with a jerk. Supination and pronation of the forearm are good. The left infra-spinatus, deltoid, biceps, brachialis anticus, and supinator longus are completely atrophied (see photographs 1, 2 and 3) and react neither to faradism nor galvanism. The trapezius, the rhomboids, the supra-spinatus, the pectoralis major, the serratus magnus, the triceps and the muscles of the left forearm, other than the supinator longus, act well, as also does the diaphragm. The right grasp is 110 (average normal about 155), and the left 35 (average normal about 145). The other muscles tested give normal reactions. His gait is probably unaffected ; he may possibly bring his right heel rather more heavily down on the ground than the average person. He can " toe and heel " a line quite well. He stands steadily with his feet close together ; if he shuts his eyes he sways slightly, but little, if any, more than the average healthy person. Co-ordination of movement generally is excellent. The unatrophied muscles are extremely irritable to direct percussion.

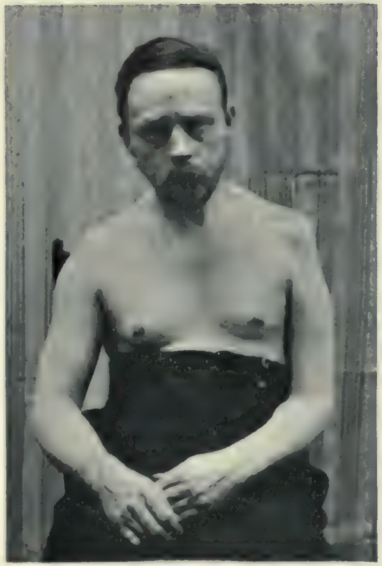


Fig. 1.

B. C.—Showing atrophy of the left deltoid, biceps, and supinator longus.

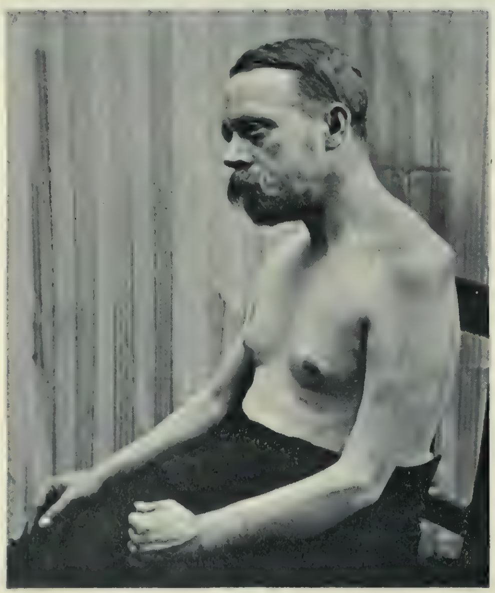


Fig. 2.

B. C.—Showing atrophy of the left deltoid, biceps, and supinator longus, also less clearly of the left infra-spinatus.

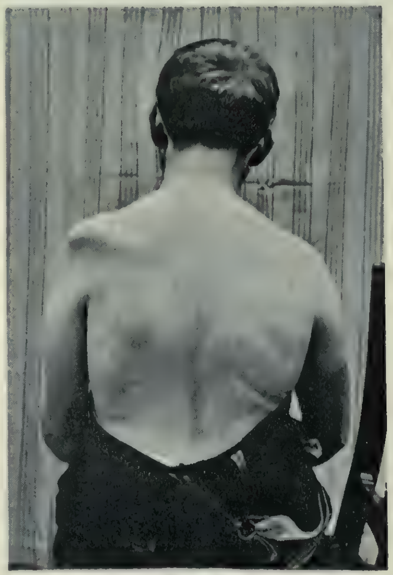


Fig. 3.

B. C.—Showing atrophy of the left infra-spinatus and deltoid.

The lightning pains are localised to the front of the thighs, the knees and the upper limbs. The girdle sensation is not constant. There is no paræsthesia or numbness. The sensibilities to touch, pain, heat, and cold are perfectly acute all over the body. Forcible compression of the ulnar nerves at the elbows causes only slight tingling in the little fingers. Sense of position is good at all the joints. There are no obvious vasomotor or trophic changes other than the muscular atrophy, except very slight grating on passive movement of the left shoulder-joint. The superficial reflexes generally are very brisk. The knee jerks cannot be elicited; the wrist jerks are fair; the right biceps-jerk is good, the left is absent. Neither the triceps jerks nor the jaw jerk are present. Except for slight tardiness of micturition the sphincters are unaffected. Smell and taste are good on each side. Hearing is about $\frac{10}{40}$ with each ear; a tuning fork held in the middle line of the head is heard equally well on the two sides, and with each ear air conduction is better than bone conduction. Vision is good, L. $\frac{6}{6}$, R. $\frac{6}{8}$; the visual fields are not restricted; colour sensation is good with each eye; the optic discs are of a good pink colour. The ocular movements, including convergence, are good. The pupils are small and unequal; the right measures 2 mm. in diameter, and the left 2·5 mm.; they do not react to light; they contract readily on convergence; they do not dilate when a painful stimulus is applied to the skin. The muscles of mastication act well, and the sensibility and movement of the face are good. The palate moves well and equally on the two sides. The larynx is normal (Sir Felix Semon). The tongue is protruded slightly to the right; it is not atrophied. There is nothing abnormal in the cranium or spine; the body organs are healthy, and the urine is normal.

This paralysis of the upper arm type obviously is not due to any peripheral lesion, because the paralysed muscles are supplied by several different nerves, which nerves supply additional non-paralysed muscles; moreover, there is no anæsthesia, although there are lightning pains both in the arms and in the legs, indicating an irritative process affecting the sensory tract. Having regard to the distribution of the atrophy, it may be held to be due to a lesion either of the anterior nerve-roots or of a group of nerve cells in the anterior horn of the cervical spinal cord.

It is probably not an anterior root-paralysis because with complete atrophy of some of the muscles supplied by the fifth and sixth cervical roots there is little if any weakness of other muscles supplied by the same roots; and results of autopsies in cases of tabes with muscular atrophy agree in showing that the anterior roots are very slightly affected, if at all, when the peripheral motor nerves are greatly changed, and even when in the same case both they and the corresponding anterior horn-cells show profound alterations.

Case II.—The second case is that of a man, D. E., aged 38, who was quite well until April, 1895, when he began to have constant numbness of his right thumb and index finger. Six months later his right little finger drooped and he was unable to straighten it. Two weeks after he noticed that his right thumb was weak and that the muscles of the ball of the thumb were smaller than previously. About the end of the December of 1895 he commenced to suffer from shooting pains in both sides of the back of the head, and a few weeks later in the left arm. In March following (1896) he found difficulty in getting his left hand up to his head when he wished to comb his hair; he had "terrible shooting pains" in the left shoulder, and he noticed a falling away of the muscle of the shoulder, of the front of the upper arm, and to a slight extent of the outer side of the forearm. Six months later (October, 1896) his right hand dropped at the wrist and at the same time he began to have pains in the right knuckles and numbness affecting the whole hand. About this time, too, he noticed an area of numbness on the side of the chest in the region of the left lower axilla. He has of late occasionally had a sensation of "drawing together" around the waist. For nine months virile power has been absent; on three occasions he has had incontinence of urine, but he has never had any trouble with the rectal sphincter. There has never been any diplopia, or difficulty in walking, or crises, or joint changes. His previous health has been excellent, except that eighteen years ago he had an attack of gonorrhœa; there were no secondary symptoms. He has always been temperate. There is no history of gout or rheumatism and there has been no special contact with lead. There is nothing of importance in the family history.

He is a moderately well-nourished man with fairly bulky but

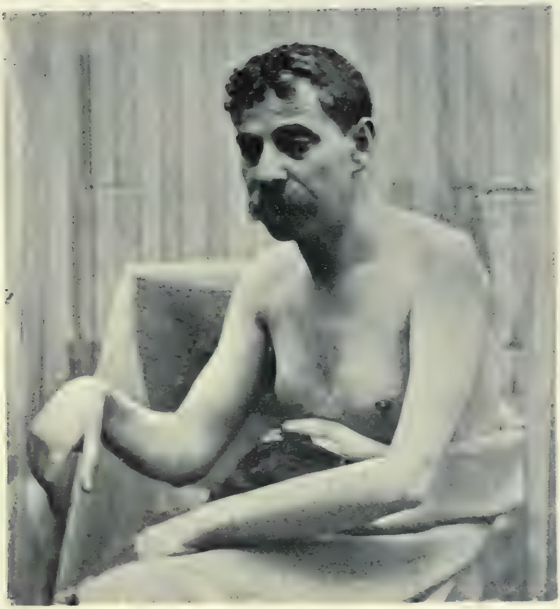


Fig. 4.

D. E.—Showing atrophy of the left deltoid, biceps, and supinator longus, also right wrist drop.

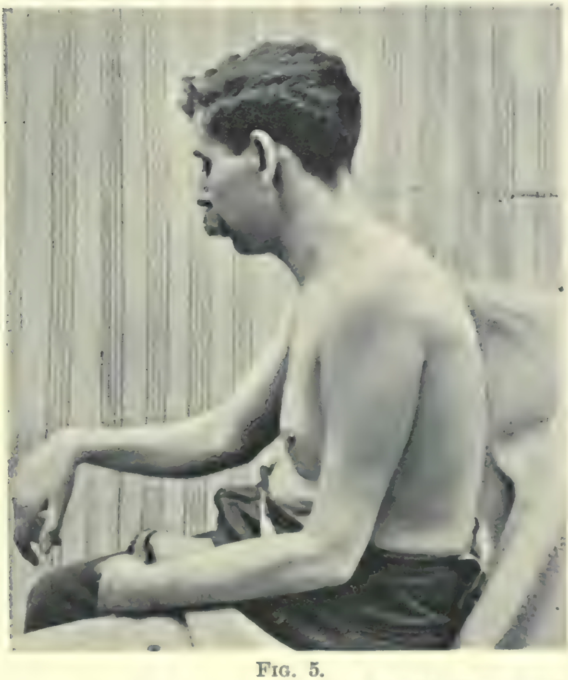


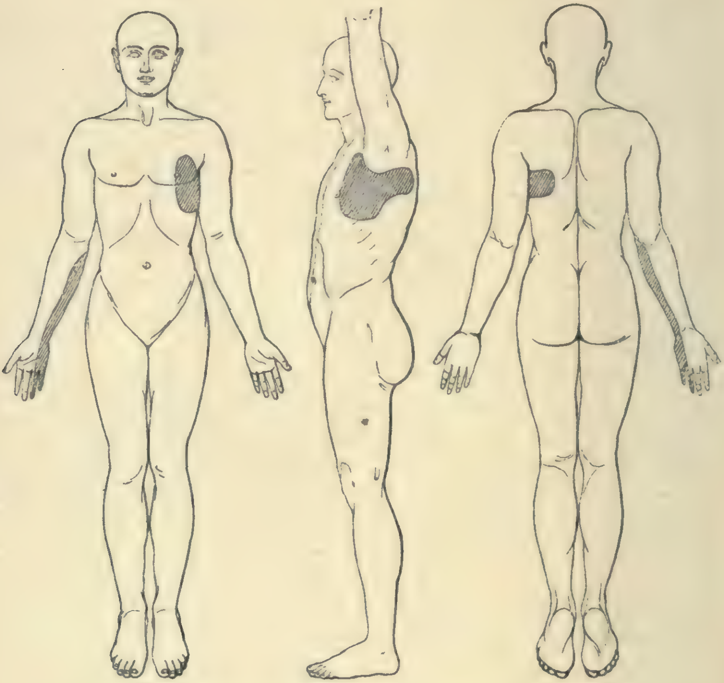
Fig. 5.

D. E.—Showing atrophy of the left deltoid, biceps, and supinator longus (and none of the infra-spinatus), also atrophy of the muscles on the extensor surface of the right forearm.

distinctly flabby muscles. His height is 5 ft. 5 in., and his weight 9 st. 7 lbs. His expression is anxious and depressed. There is no evidence of anæmia. His hair is streaked with grey. His pulse is regular but small and of low tension; its rate is 81 per minute. His mental state is excellent, except that he is inclined to be, perhaps, rather more despondent than circumstances warrant. His speech and articulation are unaffected. He is a left-handed man. There is right wrist-drop. All the muscles of the right thenar eminence are weak and they are all wasted, but more especially the adductores pollicis; he cannot touch the base of his little finger with the tip of his thumb. The hypothenar muscles are strong. The interossei act feebly. The long extensors of the fingers are paralysed and atrophied, but the extensor ossis metacarpi pollicis contracts feebly on volition. The extensors of the wrist are weak. The other muscles of the right upper limb act powerfully and are of good bulk. The intrinsic hand muscles give a reduced reaction to faradism, and the long extensors of the digits do not give any, while the extensor ossis metacarpi pollicis and the extensors of the wrist react well. To galvanism all these muscles give a reduced but brisk response with KCC>ACC.

He cannot abduct the left humerus and he flexes the elbow only very feebly, and that by means of the long flexors of the wrist and fingers alone. Pronation and supination of the forearm are good. When the elbow is extended the joint is locked with a jerk. There is complete atrophy of the deltoid, biceps, brachialis anticus, and supinator longus (see photographs 4 and 5), and these muscles do not react either to faradism or galvanism. The supra-spinatus, infra-spinatus, trapezius, the rhomboids, the pectoralis major, the serratus magnus, the triceps, the coraco-brachialis and the diaphragm act quite strongly. The gait is not at all ataxic; and in spite of his muscular weakness he can dress himself and button his own clothing.

From time to time he has (1) "gnawing" or "drawing" pain in both arms, lasting for a few minutes; (2) stabbing, shooting pains in the shoulders, back, and upper limbs, and more on the right side than the left; (3) momentary drawing-pain in the back and in the right side of the chest in the region of the lower axilla; and (4) shooting pains in the lower limbs. There is subjective numbness of the whole of the right hand and of the lower axillary region of the left side of the chest. The ground feels natural to his feet. He occasionally has girdle sensation about the level of the navel. There is anæsthesia to light touches along the ulnar border of the right forearm and hand, affecting the whole of the little finger and the ulnar half of the ring finger. There is also over this region (shown in the diagram) blunting of sensibility to painful and thermal stimuli. A similar loss of sensibility exists over an area (shown in the diagram) in the left lower axilla, extending to the mammary line in front and nearly to the angle of the scapula behind. While the left ulnar nerve is sensitive to pressure applied at the elbow the right is not. Sense of position is good at all joints.



D. E.—Loss of sensibility to touch, pain, heat and cold.

The plantar reflexes are brisk, the conjunctival a little sluggish, and the pharyngeal very brisk. The knee-jerks are absent, as also are the elbow and wrist jerks. The organic reflexes are unaffected. There are no crises or joint changes or other trophic or vaso-motor alterations. The special senses are quite acute. There is no colour blindness. The optic discs have a definite outline and a pink colour. The ocular movements are good, except convergence which is not well maintained with the right eye. There is no diplopia or ptosis. The pupils are unequal (right 6 mm., left 4 mm.), but they are regular in outline. They do not react to light, only feebly on accommodation, and they do not dilate on painful stimulation of the skin. The cranial nerves are otherwise healthy. The tongue is not atrophied. The cranium and spine show no abnormality. The body organs are healthy.

With the exception of the infra-spinatus the same upper-arm group of muscles are atrophied as in the former patient, but in this case the weakness in the first instance was associated with "terrible shooting pains in the left shoulder." Unlike the former case, there is loss of sensibility in the neighbourhood of the atrophy, but while the atrophy corresponds with the fifth cervical segment, the loss of sensibility corresponds with the fifth dorsal segment of the cord. Although one may, I think, without hesitation refer the upper-arm atrophy to a nuclear lesion, yet it is by no means so easy to form an opinion as to the origin of the atrophy in the forearm and hand of the other side. It does not correspond with an atrophy following a lesion of the ulnar nerve, inasmuch as all the thenar muscles are wasted (although the adductors of the thumb are more wasted than the rest, while the hypothenar muscles are not weak, neither is the inner half of the flexor profundus digitorum. While there is anæsthesia of ulnar distribution associated with weakness of some of the muscles supplied by the ulnar nerve, yet with atrophy of intrinsic hand muscles supplied by the median nerve there is no corresponding anæsthesia. The preservation of the extensor ossis metacarpi pollicis, while the other muscles supplied by the posterior interosseous branch of the muscular spiral nerve are atrophied (an escape not uncommon in lead paralysis), suggests a nuclear lesion. It is, therefore not improbable that the forearm and hand atrophy is not peripheral.

By the kindness of the physicians to the National Hospital for the Paralysed and Epileptic, I have been able to tabulate what I consider the important facts in relation to the subject of this paper of 200 consecutive cases of locomotor ataxia that have been under their care in the wards of that hospital during the last twelve years. In 41 per cent. there was some definite paralysis of one or more of the external ocular muscles; in 12 per cent. there was diplopia but no other evidence of paralysis; in 5·5 per cent. there were nystagmoid jerkings without obvious paralysis; in many cases with definite paralysis there was nystagmoid jerking in addition; for example, in a case where all the ocular movements were bad except the outward movement, that was accompanied by nystagmoid jerking. In 85 per cent. the movements of the iris were abnormal, usually exhibiting inactivity to light alone, but sometimes inaction both to light and accommodation.

In 28 per cent. of the cases in which the larynx was examined with the laryngoscope there was some degree of paralysis of one or both vocal cords; in 20 per cent. of these there was in addition paralysis, or in one case merely fibrillary tremor, of half the tongue on the side corresponding with the laryngeal paralysis, and also in about a half (12 per cent.) of the latter cases there was observed paralysis of the palate on the same side.

In 4·5 per cent. of all the cases in which the larynx was examined there were laryngeal crises without any obvious paralysis; in 3·5 per cent. there were ataxic movements of the cords without paralysis; and in one case there were laryngeal crises with ataxia of the cords but no definite paralysis. The laryngoscopic examination in nearly all of these cases was made by Sir Felix Semon.

In 80·5 per cent. of the total number of cases there was vesical incontinence, or laboured micturition, or both; thus in 61·5 per cent. there was incontinence of urine, and in several of these cases it was noticed that sensation during micturition was good; in 19 per cent. there was either tardiness in starting the act, intermittent or feeble stream, or the patients complained of diffculty or strain in micturition, or of a lack of expelling power, some stating that they felt after each act that they had not completely emptied the bladder.

In 34 per cent. of all the cases there was defect associated with defæcation; in rather more than half it was of the nature of extremely obstinate constipation, in rather less than half there was definite rectal incontinence, in some cases only after the use of aperient medicines; while in several of the cases there was laxity, or gaping of the sphincter, or prolapse of the rectum, or other objective evidence of muscular insufficiency. In a few of the cases with and without gastric crises there was evidence of dilatation of the stomach, and at the autopsy in one case with crises it was found to be very greatly dilated.

In 30·5 per cent. of the cases there was demonstrable motor weakness of the skeletal muscles, usually of one or other limb, but this includes 4·5 per cent. of cases of hemiplegia. The principal facts of the more important of these cases are tabulated below. In this statement all cases with weakness that could not be verified objectively while in the hospital are excluded. In many cases excluded there was a history of transitory paralysis; and in many a complaint was made of sensations of heaviness during muscular exertion, of inability to walk far, of being easily tired, and occasionally of sudden loss of power in the legs. In a considerable number of the cases, although motor power was noted to be good yet the movements of the limbs were jerky. In about 6 per cent. of all the cases it was ascertained that the patients suffered from sudden involuntary movements during severe lightning pains. These usually affected the legs, but sometimes the arms, and in one case there were spasmodic contractions of the abdominal muscles in association with marked girdle pain.

Thus in 200 consecutive cases of tabes there are sixteen with obvious atrophy, or 8 per cent., the precise percentage given by Duchenne. It is probable, however, that in several of the cases without obvious atrophy the paralysed muscles were wasted. In the table of cases with paralysis but no visible atrophy, the only paralysis which is, as it seems to me, certainly not nuclear, is that of the right side of the face with loss of taste (Case 1), and in that case the paralysis of the vocal cords with the

TABLE OF CASES OF TABES DORSALIS

No.	Name.	Age.	Duration.	Weakness or Paralysis.	Atrophy.	Electrical Reactions.
1	W. S.	34	6 yrs.	Left orbicularis palpebrarum; left external and superior recti; both masseters, temporals and pterygoids; left orbibularis oris; left half of tongue; soft palate; abductors of vocal cords; all muscles of neck; transitory of legs (for half-an-hour, with onset)	(Fibrillary twitching of orbicularis oculi on both sides); masseters and temporals of both sides; muscles of neck generally.	Orbiculares oculi react feebly to faradism and give A C C > K C C; no reaction in muscles of mastication; R. D. in left orbicularis oris; no reaction to faradism from soft palate; splenius capitis of both sides reacts feebly to faradism, the other neck muscles react well
2	C. J.	32	1½ yrs.	External eye muscles; orbiculares palpebrarum; orbiculares oris; all muscles of left face; right temporal and masseter	Almost complete of right temporal and masseter	Reduced response to faradism in muscles of left face; right orbicularis oculi; right orbicularis oris; right masseter and temporal
3	E. L.	39	2 yrs.	External ophthalmoplegia; abductors and internal tensors of vocal cords; right half of tongue (difficulty in swallowing); extensors and flexors of knees; abductors and adductors of thighs; diaphragm	Right half of tongue	Normal
4	B. C.	33	2 yrs.	Right external rectus; left half of tongue; abductors and internal tensors of vocal cords	Left half of tongue (anterior); deltoid and "arm muscles"	Normal
5	H. C.	37	2½ yrs.	Palate; left vocal cord; left trapezius; left sternomastoid	Left trapezius, with fibrillary twitchings	Left sternomatoid no reaction to faradism; L. trapezius feeble; to galvanism they give a sluggish response with A C C > K C C
6	E. W.	62	1 yr.	.. ..	Thenar muscles R. > L.; R. Extensors of wrist and fingers; R. Flexors of wrist and fingers; R. Triceps; R. Biceps, deltoid (posterior part); R. Supra spinatus, infraspinatus; R. Thigh muscles; muscles below both knees	Reduced in all but K C C > A C C; no response to faradism from R. quadriceps extensa; R. Hamstring muscles; muscles below R. knee
7	T. H. C.	59	3 yrs.	Almost complete paralysis of both upper limbs; also both lower limbs, the hamstrings alone can be feebly contracted	Muscles of upper limbs, including deltoids and pectorals, but especially intrinsic muscles of hands; muscles of lower limb	Reduced response from wasted muscles but K C C > A C C
8	D. J.	52	12 mths.	Deltoids; flexors of forearms	Deltoids; scapular muscles	Reduced in the wasted muscles
9	M. E.	28	2 yrs.	Both hands, L. > R., especially of ulnar fingers	Intrinsic muscles of hands L. > R. (fibrillary twitchings in flexors of forearms)	.. ..
10	W. W.	52	12 yrs.	Bi-lateral paralysis of abductors and internal tensors of larynx; arms weak	Of muscles of forearms and hands, especially the interossei	.. ..

WITH MUSCULAR ATROPHY.

Ataxy.	Pain.	Sensibility.	Crises.	Trophic Changes.	Bladder weakness.	Rectal defect.
Ataxic gait; slight ataxy of hands	Shooting pains in legs	Blunted over face, soft palate, right side of the tongue, and in patches over the whole body, especially the lower limbs; muscular sense very defective in legs, less so in arms	Gastric; rectal; laryngeal	None	Tardy micturition; occasional incontinence	None
Ataxic gait; slight ataxy of arms	None	Patchy loss over face and limbs	None	None	Occasional retention and incontinence	None
Marked in upper and lower limbs	Shooting pains in legs	Patchy loss below second ribs and of ulnar borders of forearms and hands	Rectal; laryngeal	None	Tardy micturition; occasional incontinence	Occasional incontinence
Of upper and lower limbs	Shooting pains in legs and arms	Slight loss in legs and trunk	None	None	Incontinence	Incontinence
Marked of legs; slight of arms; Rombergism	Shooting pains in left leg	Patchy loss below second rib (especially on left lower limb) and on ulnar side of upper limbs; muscular sense lost from toes and left ankle	None	None	Tardy micturition	None
?None	None	Loss over right arm and both legs	None	Affection of right shoulder joint	Occasional incontinence	None
.. ..	Shooting pains in legs	No loss, but paræsthesiæ of hands and arms—"pins and needles," "rawness"	None	None	Difficult micturition	Obstinate Constipation
Ataxic gait; Rombergism	Shooting pains in legs	Normal	None	None	Difficult micturition	None
Marked of arms; slight of legs	"Rheumatic" pains in R. knee and leg	Loss along ulnar borders of arms and across the chest	None	None	Occasional incontinence	None
Of legs and of hands	Shooting in legs	Slight delay of tactile sensibility; slight loss of muscular sense in right fingers	Gastric; laryngeal	None	Incontinence	Incontinence; prolapse of rectum

TABLE OF CASES OF TABES DORSALIS

No.	Name.	Age.	Duration.	Weakness or Paralysis.	Atrophy.	Electrical Reactions.
11	D. B.	35	22 mths.	[Painless cramp in ulnar muscles of flexor surface of left forearm and of muscles of both legs; spontaneous movements of fingers]	Slight of left forearm muscles; of calf muscles with fibrillation	.. ..
12	K. E.	44	6 yrs.	Ophthalmoplegia; all movements of lower limbs weak but especially flexion of hips	Of L. thenar muscles and of the interossei of L. hand; also (?) of muscles below knees	Left thenar muscles react very badly to faradism; muscles below knees gives slight diminution
13	L. T.	40	11 yrs.	Slight left ptosis; weak abduction of thumbs; dilatation of stomach (contents clear)	Lateral part of thenar muscles wasted	.. ..
14	S. G. M.	46	10 yrs.	Weakness of right side of tongue; weakness of all movements of lower limbs	Extreme in all muscles of lower limbs	Reduced faradic excitability in all muscles of lower limbs
15	B. T. W.	52	2 yrs.	Double ptosis; weakness of both legs L. > R.; paralysis of dorsiflexors of left ankle	Of all muscles of legs	.. ..
16	W. W.	37	2½ yrs.	Double ptosis; sudden paralysis of right thigh and leg; right foot drop	Of right anterior tibial and peronei, also in less degree the other muscles of right lower limb	R.D. in the right peronei, anterior tibial muscles and hamstrings

right half of the tongue and the transitory paralysis of the hand are quite probably nuclear. The paralysis of the palate, tongue and vocal cords (in Case 4) is almost certainly nuclear. In the remaining cases the origin of the atrophy is, I think, uncertain. In the table of "cases with atrophy" the weakness of the orbicularis palpebrarum (in Case 1) associated with paralysis of some of the external eye-muscles, and of the left half of the orbicularis oris associated with paralysis of the left half of the tongue, are almost certainly nuclear; also the ophthalmoplegia with weakness of the orbiculares palpebrarum in Case 2; and the atrophy of the deltoids and scapular muscles with paralysis of the flexors of the elbow joints. It is probably not possible to form a confident opinion as to the nature of the atrophy in the remaining cases, but it is likewise probably not possible to exclude a nuclear origin in any.

WITH MUSCULAR ATROPHY.

Ataxy.	Pain.	Sensibility.	Crises.	Trophic Changes.	Bladder weakness.	Rectal defect.
Jerky movements of legs	Shooting in legs; pain in legs with defæcation	Loss over ulnar border of left forearm; hyperæsthesia of back; muscular sense in limbs impaired	[Spasmodic cough and dyspnœa following tingling over larynx]	None	Incontinence	None
None	Shooting in legs	Blunting with loss of muscular sense in both lower limbs	None	None	Tardy micturition	Obstina constipation
Marked of legs; slight of arms	.. ..	Tactile anæsthesia up to level of third rib	?	None	Incontinence	Obstina constipation
Marked in arms (cannot stand alone, if supported); gait very ataxic	Shooting in legs and arms	Lost over lower limbs and trunk; impaired in hands; muscular sense in legs lost	Gastric	None	Incontinence	Incontinen
Gait wildly ataxic	Shooting in legs	Anæsthesia of feet and perinæum; analgesia of lower limbs	None	None	Micturition difficult	None
Gait ataxic; flourishes right foot	None	Patchy anæsthesia of right leg; analgesia below level of second rib and along ulnar borders of arms	Gastric	None	Incontinence	None

THE INFLUENCE OF AFFERENT IMPULSES ON MOTOR MECHANISMS.

The well-known experiment of the "brainless frog" proves that after destruction of the cerebral centres there still remain some motor mechanisms in the spinal cord that may be excited to action by strong afferent stimuli, such as those produced by applying an acid fluid to the skin. These mechanisms represent combined movements, for the movements produced are quasi-purposive in character; and, moreover, their activity, it is to be presumed, is independent of nerves which habitually carry impulses that result in sensation. Both Sherrington[49] and Page May[50] have showed the remarkable fact that stimulation of the posterior roots (in the dog and monkey) causes contraction of groups of muscles. Page May found likewise that similar movements were produced by faradic stimuli

TABLE OF CASES OF TABES

No.	Name.	Age.	Duration.	Paralysis.	Ataxy.	Pain.
1	B. J.	39	4 yrs.	Abductor paralysis of vocal cords; weakness of right half of tongue; paralysis of right face (with loss of taste); transitory paralysis of left hand; weakness of legs, sudden in onset	Gait very ataxic; hands slightly ataxic; Rombergism	Shooting pains in lower limbs
2	D. W.	43	4 yrs.	Paralysis of left vocal cord; paralysis of right masseter and pterygoids; paralysis of muscles supplied by both third nerves	Slight of arms and legs; Rombergism	Shooting pains in lower limbs
3	T. J.	57	1 yr.	Abductor paralysis of right vocal cord; double ptosis and weakness of all ocular muscles except inferior oblique which overacts	Marked in all limbs; Rombergism	Shooting pains in legs
4	C. J.	39	1½ yrs	Weakness of all muscles supplied by both third nerves; abductor paralysis of vocal cords; weakness of right half of soft palate; weakness of right half of tongue; weakness of right side of face	Gait very ataxic	Shooting pains in legs
5	T. W.	44	13 mths.	Cannot move feet at ankles; moves toes feebly; movement at knee joints feeble; weakness of flexors of left wrist and fingers	Slight of legs and arms	None
6	G. R.	36	3 yrs.	Weakness of lower limbs, especially of left ankle; clawed feet (spontaneous athetoid movements of fingers of right hand)	Gait ataxic; also right arm; Rombergism	Shooting pains in legs
7	B. H.	41	3½ yrs.	Weakness of all movements of lower limbs, especially flexors of hips (legs suddenly "gave way" has not walked or stood since)	Movements of lower limbs unsteady	Shooting pains in legs and simultaneously in lower abdomen, causing legs to draw up
8	R. E.	41	5 yrs.	Paralysis of dorsiflexors and evertors of left foot	Marked of legs; slight of arms	Generally distributed shooting pains
9	G. F.	42	20 mths.	Weakness of left anterior tibial group of muscles (faradic excitability reduced)	Gait slightly ataxic	Shooting pains in legs
10	H. M.	45	4 yrs.	Paralysis of dorsiflexors and evertors of left foot; weakness of left lower limb [R.D. in left tibialis anticus, ext. com. dig., ext. long hall. Reduced faradic excitability in the peronei.]	Ataxy of leg; Rombergism	Shooting pains in legs, in trunk, and occasionally in arms
11	L. C.	53	5 yrs.	Weakness of dorsiflexors and evertors of right foot, also weakness of flexors and extensors of toes; weakness of evertors of left foot [right club foot]. (Faradic excitability much reduced in peroneus longus)	Ataxy of legs and hands	Shooting pains in legs with spasmodic flexion of knees; (movements of legs and right arm during sleep)
12	P. S.	54	6 mths.	Flaccid paralysis of both legs; double foot drop [no R.D.]; weakness of left external rectus	None	Shooting pains in legs; aching in coccygeal region
13	C. S.	44	6 yrs.	Weakness of dorsiflexors of left ankle	Of legs and arms; Rombergism	Shooting pains in legs

DORSALIS WITH PARALYSIS.

Sensibility.	Crises.	Trophic Changes.	Bladder.	Rectum.
Patchy loss below second rib; muscular sense impaired in lower limbs	Gastric; laryngeal	Charcot's joint; changes in both knees	Tardy micturition; incontinence	None
Loss over face	None	None	Tardy micturition; incontinence	None
Blunting in hands, feet and ulnar side of forearms	None	Perforating ulcer of foot	Tardy micturition	None
Patchy loss below second rib; loss over ulnar side of upper limbs; muscular sense lost in legs and little fingers	Gastric; laryngeal	None	Occasional retention and incontinence	None
Blunting in hands; muscular sense lost in legs and hands	None	None	Difficult	Incontinence (after aperients)
Blunting over lower limbs and right hand	None	None	None	None
Slight anæsthesia of legs	None	None	Retention; precipitate micturition	None
None	None	None	None	None
Blunting below knees	Gastric; laryngeal	None	Occasional incontinence	Occasional incontinence
Blunting below left knee; muscular sense defective in left great toe	None	None	Occasional incontinence	None
Loss over forearms, hands, and below middle of thighs	None	None	Occasional incontinence	None
Of lower limbs; of root distribution	Rectal	None	Incontinence	Tenesmus; gaping of sphincter ani; no anal reflex
Relative analgesia in arms	Gastric	None	Difficulty in micturition	None

applied to the surface of the cord in the region of the postero-external column (but not in the region of the postero-internal), and he showed that, although both flexor and extensor muscles were thrown into contraction, the "resultant" movement was one of flexion. On stimulating the postero-external columns of the lower lumbar segments he noticed rumbling of the bowels with closure and protrusion of the anus. It is possible that the spasmodic flexion of the lower limbs occurring in association with severe lightning pains may be analogous to the movements experimentally produced. Not only, however, does stimulation of the posterior roots result in movement, but conversely destructive lesions of the posterior roots produce paralysis. Claude Bernard[53] divided the posterior roots in frogs and dogs and demonstrated an ensuing paralysis of the corresponding limbs. The same phenomena of paralysis have been shown by Sherrington and Mott[48] to obtain in the monkey, and they proved that the paralysis depended more on the interruption of impulses coming from the skin (and particularly that of the palm and sole) than of those coming from the muscles. Tissot and Contejean[54] have made similar experiments with similar results; in one after section of the four lower lumbar and two upper sacral roots on one side there was not only complete paralysis of the posterior limb on that side, with absolute muscular relaxation, but also persistent incontinence of urine. In another experiment, after extirpation of the inter-vertebral ganglia of the same posterior roots, there was complete paralysis of the limb for four or five days, then the animal began again to use it, but with marked ataxy and pronounced overaction of the extensor muscles. In explanation of these results Sherrington and Mott suggest that the interruption of sensory impulses passing to the cortex cells is the cause of the paralysis; but, as Bastian[52] states, in those comparatively rare cases where the upward prolongations of the posterior root neurons are destroyed in the hinder part of the internal capsule, there is no paralysis, and he suggests that, as in lesions of the commissure between the auditory word-centre and the motor speech-centre, there results an aphasia as absolute as that produced by lesions of Broca's convolution itself; so, after interrupting the afferent impulses that normally go by the reflex collaterals to the anterior horn-cells, there ensues a molecular change in these cells which blocks the passage of "voluntary" impulses from the cortex to the muscles. It is not difficult to find clinical phenomena that may correspond in their mechanism with the changes artificially produced; for example, the giving way of the legs in association with severe lightning pains, which, as Buzzard and others have stated, is truly a paralytic effect, the weakness of the sphincters, the inability to empty the bladder, the obstinate constipation, and the laxity of the sphincter ani.

That the muscle tonus is abolished by section of the posterior roots is a fact established by the experiments quoted, and as is well known (particularly through the observations of Fraenkel[55]), in tabes the tonus of the muscle is so much diminished that the limbs of the patient may be moved passively, without pain, into attitudes impossible in health; although at the same time it should be remembered the mechanical irritability of the muscle is greatly in excess of the average normal.

In addition to the loss of tonus, Mott and Sherrington[48] observed "a certain degree of wasting" of the paralysed muscles but "no appreciable alteration of colour"; Tissot and Contejean likewise found the muscles slightly atrophied. From the phenomena of the so-called arthritic atrophy one may infer that an irritative lesion of afferent nerves is more effective in producing atrophy than a paralysing one, for Raymond[56] showed that after the injection of an irritant, such as a solution of a mercury salt, into the knee joints, atrophy of the surrounding muscles ensued unless the posterior roots were divided. Massary,[56] following the suggestion of Dejerine, believes that the muscular atrophy of tabes corresponds essentially with that seen in hemiplegia; that for the exercise of the normal trophic energy of an anterior horn-cell on its neuron it is necessary that afferent impulses should reach it, on the one hand, from the cortex by a pyramidal fibre, and, on the other, from the peripheral nerve ending in the skin, by the posterior root and reflex collateral; that when either channel is interrupted the nutrition of the cell suffers and muscular atrophy results. It is quite true that in some cases of hemiplegia there is marked atrophy, but one can hardly admit it to be comparable with that seen in tabes, chiefly because there is no loss of muscular tonus, or any approach to the reaction of degeneration, while the atrophy is never absolute so as to abolish galvanic excitability as may occur in tabes.

Observations on the changes that result in the anterior horn-cells after division of the posterior roots have recently been made by Warrington,[51] who found distinct evidence of chromatolysis of the cells, mainly of those of the postero-external group in the lower cervical and lower lumbar regions.

Schaffer,[44] as previously remarked, found similar changes after using Nissl's method in a case of tabes with atrophy of the peroneal muscles. Charcot[7] long ago traced, in a case of tabes with club-foot, marked degenerative changes from the posterior roots along the reflex collaterals to the anterior horns. Condoleon[26] particularly emphasised the fact that profound changes may exist in the anterior horn-cells together with marked degeneration of the intra-muscular motor nerves, and yet the larger nerve-trunks and the anterior nerve-roots be almost unchanged. As Schaffer points out, the failure of Dejerine to find cell degeneration in the presence of peripheral lesions loses its value as evidence because the observations were made before the more recent methods of demonstrating slight structural alterations in nerve cells were invented. It follows, then, that in some of the cases of muscular atrophy in tabes the lesion is nuclear, and in others it is not at present possible to exclude either a central or a peripheral lesion; and Dr. Hughlings Jackson's views, to which reference was made at the beginning of this paper, are confirmed by the discovery of chromatolysis of the anterior horn-cells. It would seem, therefore, that tabes is as much a segmental disease as a system disease, that it is indeed strictly a neuron disease.

I desire in conclusion to express my gratitude to the physicians of the National Hospital for the Paralysed and Epileptic for the generous manner in which they have permitted me to make use of the notes of their cases, and more especially to Dr. Ferrier for his kindness in allowing me to publish the two cases here recorded.

REFERENCES.

[1] CRUVEILHIER, J., *Anatomie pathologique du Corps Humain*, tom. i., liv. 32, p. 19.

[2] ERB, W. H., *Von Ziemmsen's Cyclopædia*, vol. xiii., p. 572.

[3] LABORDE, "Note sur un cas d'atrophie et de dégénérescence amyloïde de la moëlle épinière ; paraplégie ; atrophie et ataxie musculaire," *Soc. de Biologie*, 1859, p. 184.

[4] DUCHENNE, G. B., "L'Electrisation localisée," 1861, p. 603.

[5] DUMENIL, "Note sur la dégénérescence, avec atrophie, des cordons postérieurs de la moëlle épinière, avec l'ataxie locomotrice progressive," *L'Union Medicale*, tome xiii., 1862, No. 17, p. 261.

[6] FRIEDREICH, N., "Ueber degenerative Atrophie der Spinalen Hinterstränge," *Virchow's Archiv*, bd. xxvii., s. 25.

[7] CHARCOT, J. M., *Compt. rend. de la Soc. de Biol.*, 1871, p. 9.

[8] PIERRET, M. A., "Sur les altérations de la substance grise de la moëlle épinière dans l'ataxie locomotrice considérés dans leur rapports avec l'atrophie musculaire qui complique quelquefois cette affection," *Archives de Physiol. normale et pathologique*, 1870, p. 599.

[9] JEAN, A., "Ataxie-locomotrice progressive, troubles ataxiques du côté du larynx et du pharynx," *Le Progrès Medical*, 1876, No. 20, p. 379.

[10] CUFFER, "Sur un cas de sclérose des cordons postérieurs ; ataxie locomotrice, avec atrophie de la main droite et hémiatrophie droite de la langue," *Compt. Rend. de la Soc. de Biol.*, 1875, p. 249.

[11] RAYMOND ET ARTAUD, "Note sur un cas d'hémiatrophie de la langue survenue dans le cours d'un Tabes dorsal," *Arch. de Physiol.*, 1884, p. 367.

[12] KAHLER, O., "Beitrag zur pathologischen Anatomie der mit cerebralen symptomen verlaufenden Tabes dorsalis," *Zeitzch. f. Heilkünde*, 1881, bd. ii., s. 432.

[13] BUZZARD, T., "On Ophthalmoplegia Externa in conjunction with Tabes Dorsalis, with some remarks on Gastric Crises," BRAIN, vol. v., April, 1882, p. 34.

[14] BUZZARD, T., "On certain little recognised phases of Tabes Dorsalis (Locomotor Ataxy)," *Lancet*, September 24, 1881, p. 539.

[15] LANDOUZY ET DEJERINE, "Recherches sur les altérations bulbaires chez les ataxiques à crises laryngées," *Compt. Rend. de la Soc. de Biol.*, 1883, p. 367.

[16] EISENLOHR, "Ueber bulbäre Complicationen der Tabes," *Deutsche Med. Woch.*, 1884, s. 554.

[17] BALLET, G., "De l'hémiatrophie de la langue dans le Tabes dorsal ataxique," *Arch. de Neurologie*, 1884, p. 191.

[18] Koch, P. D., et Marie, P., "Contribution a l'Etude de l'Hémiatrophie de la Langue," *Revue de Médec.*, viii., 1888, p. 1.

[19] Fournier, A., *Leçons sur la Période préataxique du Tabes d'origine syphilitique*, Paris, 1884, p. 105.

[20] Eulenburg, A., "Ein Fall von Tabes dorsalis complicirt mit progressiver Muskelatrophie," *Berl. Klin. Wochensch.*, April 13, 1885, p. 229.

[21] Hacker, F. H., "A case of Tabes Dorsalis complicated with progressive muscular atrophy," *Med. News, Philad.*, 1885, p. 288.

[22] Pitres, A., et Vaillard, L., "Contribution a l'étude des névrites périphériques chez les tabétiques," *Rev. de Méd.*, vi., 1886, p. 574.

[23] Fischer, G., "Ueber vorübergehende Lähmung mit Ea. R. im Prodromalstadium der Tabes," *Berl. Klin. Wochen.*, 1886, s. 561.

[24] Remak, E., "Ueber neuritische Muskelatrophie bei. Tabes dorsalis," *Berl. Klin. Woch.*, 1887, s. 462.

[25] Westphal, C., "Ueber einen Fall von chronischer progressiver Lähmung der Augenmuskeln (ophthalmoplegia externa) nebst Beschreibung von Ganglienzellengruppen im Bereiche des Oculomotoriuskerns," *Arch. f. Psychiat. und Nervenkrank*, xviii., 1887, s. 846.

[26] Condoléon, J., *Contribution a l'étude pathologénique de l'amyotrophie tabétique*, Paris, 1887.

[27] Joffroy, A., "Du pied bot tabétique ; atrophie des muscles de la jambe," *Bull. de la Soc. Médicale des Hopitaux*, 1885, p. 446.

[28] Braun, H., "Ueber einen eigenthümlichen Fall von combinirter systematischer Erkrankung des Rückenmarks und der peripheren Nerven," *Deutsch. Arch. f. Klin. Med.*, Leipzig, 1888, s. 461.

[29] Hoffmann, J., "Beitrag zur Ætiologie, Symptomatologie und Therapie der Tabes dorsalis," *Arch. f. Psychiat. und Nervenkrank*, bd. xix., 1888, s. 456.

[30] Stransky, M., "Ein Fall von mit Muskelatrophie complicirter Tabes," *Prager Med. Woch.*, xiii., 1888, s. 248.

[31] Schultze (Bonn), "Kaumuskellähmung bei Tabes," *Centralb. f. Nervenk.*, Leipzig, 1889, s. 362.

[32] Dejerine, M. J., "De l'atrophie musculaire des tabétiques et de sa nature périphérique," *Compt. Rend. de la Soc. de Biol.*, 1888, No. viii., p. 194.

[33] Dejerine, M. J., "Etude clinique et Anatomo-pathologique sur l'Atrophie Musculaire des Ataxiques," *Rev. de Médecine*, tom. ix., Fev. 1889, p. 81.

[34] Dejerine, M. J., "Des Paralysies au cours du Tabes," *La Médecine Moderne*, No. xiii., 1890, p. 245.

[35] Howard, R. P., "Case of Bilateral Ophthalmoplegia externa and interna associated with Tabes Dorsalis, Bulbar Paralysis, and loss of vision and hearing," *Internat. Jour. Med. Sci.*, N.S., xcvii., 1889, p. 238.

[36] Goldscheider, "Ueber atrophische Lähmung bei Tabes dorsalis," *Zeitschr. f. Klin. Med.*, bd. xix., 1891, s. 444.

[37] Gerhard, C., "Tabes mit Zwerchfellslähmung," *Berl. Klin. Woch.*, xxx., 1893, s. 369.

[38] Ilberg, "Ueber Lähmung des XI. Gehirnnerven bei Tabes dorsalis," *Charité-Annalen*, Berlin, 1893, xviii., s. 303.

[39] Charcot, J. M., "Le Syndrome paralysie labio-glosso-laryngée progressive dans le tabes," *Le Progrès Médical*, 1893, 2e. S. xvii., p. 449.

[40] Lepine, M., "Troubles glosso-laryngés dans le tabes," *Lyon Médical*, 1894, lxxx., p. 233.

[41] Dejerine, M., et Petreen, K., "Sur un cas d'ophthalmoplégie externe totale et de paralysie laryngée relevant d'une névrite périphérique à marche rapide chez un malade atteint de Tabes au début," *Compt. Rend. Soc. de Biol.*, 1896, p. 822.

[42] Charcot, J. B., et Dufour, H., "De l'hemiarthrose tabétique et de deux symptomes rares dans le cours du Tabes dorsalis, Paralysie dans le domaine d'une nerve spinal (crural) et tremblement ataxique de la langue," *Nouvelle Iconogr. de la Salpêtrière*, tome ix., 1896, p. 266..

[43] Gowers, W. R., "Diseases of the Nervous System," 1892, vol. i., p. 417.

[44] Schaffer, C., "Sur l'origine de l'amyotrophie tabétique," *Revue Neurolog.*, Paris, 1896, iv., p. 97.

[45] Leyden, "Ueber die Betheiligung der Muskeln und motorischen Nervenapparate bei der Tabes dorsalis," *Deutsche Zeitschr. f. prack. Med.*, 1887, No. 50. (Referred to by Condoleon, p. 22, and others.)

[46] Jackson, J. Hughlings, "On the Nature of the Disorders of Co-ordination in Locomotor Ataxia," *Lancet*, Jan. 30, 1875, p. 161.

[47] Jackson, J. Hughlings, "Neurological Fragments," No. xvi., *Lancet*, Dec. 12, 1896.

[48] Mott, F. W., and Sherrington, C. S., "Experiments upon the Influence of Sensory Nerves upon Movement and Nutrition of the Limb," Preliminary Communication, *Proc. Roy. Soc.*, vol. lvii., 1894-95, p. 481.

[49] Sherrington, C. S., "Notes on the arrangement of some Motor Fibres in the Lumbosacral Plexus," *Jour. of Physiol.*, vol. xiii., 1892, p. 622.

[50] May, W. Page, "Investigations into the Segmental Representation of Movements in the Lumbar Region of the Mammalian Spinal Cord," *Phil. Trans.*, B. 1897, vol. clxxxviii., p. 191.

[51] Warrington, W. B., "On the Structural Alterations observed in Nerve Cells," *Jour. of Physiol.*, vol. xxiii. (Nos. 1 and 2, June 10), 1898, p. 112.

[52] Bastian, H. Charlton, "Note on the Relations of Sensory Impressions and Sensory Centres to Voluntary Movements," *Proc. Roy. Soc.*, vol. lviii., April, 1895, p. 89.

[53] Bernard, C., *Leçons sur la Physiologie et la Pathologie du Système Nerveux*, No. 14, p. 252.

[54] Tissot, J., et Contejean, C., "Sur les effets de la rupture du circuit sensitivo-moteur des muscles dans sa portion centripète," *Compt. rendus de la Soc. de Biol.*, 1895, p. 569.

[55] Frenkel, "Ergebnisse und Grundsätze der Uebungstherapie bei der tabischer Ataxie," *Neurol. Centralb.*, 1897, p. 474.

[56] Raymond, "Recherches Expérimentales sur la pathogenie des atrophies musculaires consecutives aux arthrites traumatiques," *Rev. de Médecine*, 1890, p. 374.

[57] Massary, E. de, *Le Tabes dorsalis, dégénérescence du protoneurone centripète*, Paris, 1896, p. 117.

[58] Chauveau, A., "On the Sensorimotor Nerve-circuit of Muscles," Brain, vol. xiv., 1891, p. 195.

ON TWO CASES OF EARLY SYPHILITIC PARAPLEGIA.

(*A year after primary syphilis*).

BY F. PARKES WEBER, M.D., F.R.C.P.

With Histological Report by J. S. Risien Russell, M.D., F.R.C.P.

So considerable a proportion of cases of acute spinal paraplegia give a history of primary syphilis about a year previously to the onset of the spinal disease, that one is forced to consider the paraplegia as really due to the syphilis, though *post-mortem* examination of the cord in such cases may show nothing which is absolutely peculiar to syphilis.

The following two cases deserve to be recorded, chiefly on account of the histological report kindly furnished by Dr. Risien Russell, to whom I am much indebted.

Case I.—Charles S., aged 23, was admitted to the German Hospital in June, 1896, suffering from paraplegia, bedsores, and hectic fever. Ten months ago he had a chancre, and four months later a rash appeared, the remains of which were still visible when he entered the hospital. For the six weeks previous to admission both his legs had been paralysed. He was unable to retain his urine and fæces. Sensation was absent up to about a hand's breadth above the umbilicus. Treatment by mercurial inunction and iodide of potassium did no good, and the patient died nineteen days after admission.

At the necropsy the spinal cord appeared natural or almost natural to the naked eye, and there was no obvious transverse softening to be found.

Dr. Risien Russell's Report.

In this case nearly the whole of the thoracic together with the lumbar and sacral regions of the spinal cord were available for examination. Evidences of meningitis are seen both in the thoracic and lumbar regions, but the exact degree of this, and its extent, varies at different levels. In the upper thoracic region the meninges surrounding the whole circumference of the cord show marked signs of inflammation, while they are less uniformly involved in the lower thoracic region, and still less so in the lumbar cord. The meninges in these less affected parts show little or no change in some areas, while in others the change is pronounced, the distribution of the meningitis being somewhat patchy. The changes in the meninges consist in thickening with cell proliferation and exudation ; the vessels are dilated, many of them engorged, and their walls thickened with multiplication of the nuclei.

The spinal cord itself is most pronouncedly altered in the upper thoracic region where marked myelitic changes exist ; the nerve elements are greatly destroyed and are replaced by granular material, round cell proliferation and an increase of interstitial tissue. The vessels are much engorged, their walls markedly thickened, with increase of nuclei and perivascular proliferation of round cells. Other of the vessels are completely occluded, a fibrous cord without any evidence of a lumen being met with in such cases. Precisely similar changes are met with in the grey matter, where the round cell proliferation is even more pronounced than in the white, but not so great as in the immediate vicinity of the vessels in the latter. The shape of the grey matter as seen in mass is greatly altered, the distortion of it being most marked in connection with the anterior horns. Changes similar in kind, though much less extensive in distribution, as regards the amount of the transverse area of the cord affected, are seen in the lower thoracic region ; and though the grey matter is involved as well as the white, the former preserves its characteristic shape instead of being distorted as in the upper thoracic region. Sections from the lumbar cord show still slighter changes, which can be regarded as the direct result of an inflammatory process. There is an increase of round cells in the grey matter and in the white matter of the lateral and ventral regions of the cord, and the interstitial tissue is thickened chiefly in the same parts. Many of the vessels have thickened walls, but few of them are engorged, only one or two here and there being found in this condition both in the grey and white matter.

The destructive changes of the nerve elements are, of course, most pronounced in the upper thoracic region, and here the nerve fibres have suffered in greatest degree at the periphery of the cord and in the posterior columns, one half of the cord being damaged to a somewhat greater extent than the other. Sections prepared by the Weigert-Pal method show that in the most affected parts only scattered nerve fibres remain whose myelin sheaths are stained, and some of these are obviously swollen and are undergoing various degrees of degeneration, while the sheaths of other fibres take on the stain very imperfectly. Some of the swollen fibres are darkly stained throughout their whole transverse extent, there being no distinction between the axis-cylinder and myelin sheath. In addition to these changes, large blobs of variously altered myelin are to be seen, some partly stained and some quite unstained, while there also exist large and small empty spaces from which the products of degeneration have escaped, or have been removed. The white matter which bounds the grey is better preserved than is that at the periphery of the cord; but even here changes exist which are similar in character, though considerably less in degree. Some of the degenerated fibres in the posterior columns and antero-lateral regions of the cord are no doubt to be accounted for by ascending degeneration, the result of destructive inflammatory lesions in other segments nearer the caudal end of the cord, but the major part of the degeneration of the nerve elements can well be accounted for by the presence of the local inflammatory changes which have been already described.

The degenerative changes in the white matter diminish in amount as we pass toward the caudal end of the cord, so that about the mid-thoracic region the white matter of the periphery, though still more affected than that more centrally situated, is but moderately degenerated in some parts, especially in the ventral region of the cord. The lower thoracic region is very much better preserved, so that beyond some evidence of descending degeneration in the cross pyramidal tracts and in the antero-lateral region, only islets of degeneration of nerve fibres exist, the more important of which occupy the antero-lateral region, especially on one side, while smaller areas of the same kind exist in the posterior columns. With the exception of a few degenerate fibres here and there, the lumbar cord is free from degeneration of the nerve elements other than that which occupies the cross pyramidal tract regions and the anterior columns, and which is evidently the result of descending degeneration consequent on destructive processes in the thoracic cord.

The neurons of the anterior horns are profoundly altered, the cells are completely destroyed in the upper thoracic region, where, with the exception of a very occasional much swollen and obviously degenerated cell, none are to be seen. The same may be said of the cells of Clarke's columns and of those of the posterior horns. Similar destructive changes in connection with the neurons exist in the caudal part of the thoracic cord, where, however, more of the cells have escaped total extinction, especially in Clarke's columns; but most of these are swollen and obviously degenerated, and only very exceptionally a fairly normal looking cell is seen. In marked contrast to the condition of the neurons in the thoracic region, those in the lumbar cord are well preserved; most of the cells appear quite normal, only one here and there showing any evidence of degenerative change, and that but slight as compared with what has already been described in connection with the thoracic cord.

Case II.[1]—R. W., aged 42, was admitted into the German Hospital in September, 1894. Patient had had a chancre and skin eruption a year before admission, in September of 1893. He had infected his wife, who was suffering from secondary symptoms at the time of patient's admission to the German Hospital. There was, therefore, no doubt as to the correctness of the history of syphilis. Six weeks before admission patient began to have difficulty in micturition, and some loss of power in lower extremities gradually showed itself. Three days before admission he suddenly became completely paralysed in the left leg. On admission there was complete paralysis of the left lower limb and motor paresis of the right one. Knee-jerks were absent. Sensation was very slightly if at all impaired. The bladder was completely paralysed, and had to be emptied at regular intervals. There was likewise incontinence of fæces. Two days after admission paralysis of the right lower limb became complete.

Treatment was begun on admission—mercurial inunction with 1¼ drachms of the unguentum hydrargyri daily, and iodide of potassium internally (15 grains thrice daily). After three weeks, when the patient had had eighteen inunctions, the treatment was left off, as it was found to have done no good. The paralysis remained stationary. Acute decubitis showed itself soon after admission, and this and cystitis were the ultimate cause of the patient's death six weeks after admission. There was hectic fever during the last four weeks. Tactile sensation was not lost in the paralysed limbs, but special sensation for heat and cold was not examined.

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[1] This case was described in the Discussion, "On the Affections of the Nervous System occurring in the Early (secondary) Stages of Syphilis," at the Royal Medical and Chirurgical Society in 1895 (*Proceedings*, vol. vii., p. 108), but the cord had not yet been examined microscopically.

At the *post-mortem* examination a transverse area[1] of softening was found in the spinal cord, which for about an inch was diffluent and pinkish in colour. The softened area with parts of the cord above and below it were removed for microscopic examination.

Dr. Risien Russell's Report.

Sections from the cervical and thoracic regions of the spinal cord were alone available for examination. These show evidences of meningitis; the meninges are thickened by exudation, the vessel walls are also increased in thickness with multiplication of nuclei, and many of the vessels are dilated, some being full, while others are empty, and some of the latter are flattened so that opposite sides of the inner wall of the vessels are in contact or almost so. No occlusion of any of the meningeal vessels by a process of endarteritis is to be seen. The only sections in which the meninges are sufficiently well preserved for satisfactory study of these changes are those of the thoracic region below the chief seat of softening; but there is enough evidence from the other sections to make it certain that meningitic changes also exist in the upper regions of the cord.

The sections show that the spinal cord itself is greatly destroyed in one part; much of this is the result of myelitis, but a good deal is also due to the unavoidable damage which has been done to this softened part during the process of preparation for microscopic examination. It is not possible to differentiate grey from white matter, and the general characters of the sections are so altered that it is impossible to say from what region of the cord it has been taken; but an examination of sections from parts of the cord above and below the lesion makes it clear that this chief area of softening must have existed in some part of the upper or middle region of the thoracic cord. The nerve elements are profoundly altered in this part, the myelin sheaths are seen in various stages of degeneration, and nothing resembling a nerve cell can be seen. There is extensive exudation, with marked vascular engorgement, and actual hæmorrhages in some places. In spite of these pronounced myelitic changes, however, there are many nerve fibres whose myelin sheaths have escaped destruction; in such the differentiation between myelin sheath and axis-cylinder is preserved, but even these fibres stain badly.

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[1] I was not present at the *post-mortem* examination, and at the Society was doubtless wrong in stating that the chief area of softening was in the lower dorsal region. Dr. Russell's report shows that it was in the upper or middle thoracic region.

Though the part of the cord just described is that chiefly affected, the process is not simply a transverse myelitis affecting this region and it alone. The change is a much more widespread one, a similar state of things, slighter in degree, existing in the cervical cord, and in the thoracic region below the chief seat of softening. In the cervical region, which is more affected than the lower thoracic, the change is most marked in the lateral regions of the cord, and more so on one side than on the other, while the posterior columns are only slightly affected, and there is no evidence of ascending degeneration in these tracts. Most of the vessels are engorged, and as a rule their walls are thin; but there are no hæmorrhages. Here and there a small vessel is seen with thickened walls and its lumen much narrowed; but no actually occluded vessels are seen. The grey matter of the anterior and posterior horns show similar changes: exudation, engorged vessels and even hæmorrhagic extravasations. A few of the anterior horn cells are preserved, but a great many have been destroyed, especially on one side.

In the thoracic cord below the chief seat of softening there is evidence of descending degeneration in both crossed pyramidal tracts, and in the direct pyramidal tract of one side, the opposite direct tract showing little or no change. In addition to this there is some evidence of some inflammatory change, but this is very slight, and in no way to be compared with that met with in the upper regions of the cord.

The absence of ascending degeneration in the cervical region of the cord, and the presence of descending degeneration in the lower thoracic region, make it probable that the myelitic process did not affect the posterior columns to any extent at any level below the part of the cervical cord examined; but that the chief brunt of the mischief must have fallen on the lateral and ventral regions of the cord. Moreover, the absence of descending degeneration in one direct pyramidal tract makes it clear that one ventral half of the cord must have suffered in greater degree than the other at a period sufficiently remote for descending degeneration to result.

REMARKS.

The vascular changes which have been claimed as typical of syphilitic myelitis were present to a marked degree in Case I. but are comparatively absent in Case II. This constitutes probably the most important histological difference between the two cases. In fact, in the first case the degeneration of the nerve fibres seems to be due largely to the cutting off of their blood-supply owing to the vascular affection, whilst in the second case it seems to be due chiefly to some direct action of the syphilitic poison on the nervous elements themselves. Cases of the first kind have been described by E. Lanceraux,[1] H. Lamy,[2] E. Biernacki,[3] and others. Although T. Rumpf[4] suggests that many cases of so-called acute ascending paralysis are really due to syphilitic vascular changes, even Lanceraux[5] acknowledges that syphilis is probably not the only disease which can give rise to obliterative changes in the spinal vessels, and in Biernacki's[6] first case there was no history of syphilis. On the other hand my second case shows that the vascular changes in the cord are not always a chief feature of syphilitic cases. In fact as Dejerine[7] maintains, syphilitic cases of acute spinal myelitis cannot yet be distinguished symptomatically, during life, or pathologically, after death, from non-syphilitic cases, except by the history of fairly recent syphilitic infection.

At one time it was sometimes maintained that there was no such thing as a diffuse syphilitic affection of the spinal cord without grave meningeal changes, but that all were cases of meningo-myelitis. Though undoubtedly many of the published cases are examples of this, other cases show that the syphilitic spinal affection can exist without the meninges being macroscopically much affected. In such cases the parenchymatous affection of the spinal cord is not secondary to meningitis.[1] My first case at least may be cited as an example of this.

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[1] *Vide Leçons de Clinique Médicale*, Paris, 1892, p. 217.

[2] "De la méningo-myélite syphilitique," in *Nouvelle Iconographie de la Salpêtrière*, 1893, vol. vi.

[3] "Myelopathia endoarteriitica acuta," in *Deut. Zeitschr. für Nervenheilkunde*, 1897, vol. vii., p. 173.

[4] *Die Syphilitischen Erkrankungen des Nervensystems*, Wiesbaden, 1887, p. 340.

[5] *Loc. cit.*, p. 218.

[6] *Loc. cit.*

[7] "De la myélite aigüe centrale survenant chez les syphilitiques à une époque rapprochée du début de l'affection"; in *Revue de Médecine*, Paris, Jan., 1884, p. 60 and p. 76.

It is noteworthy that in the first case no distinct macroscopic lesion could be detected in the cord. A similar fact is noted in certain other recorded cases, as in those described by W. Möller[2] and H. Schmaus.[3]

The importance of early treatment in all similar cases need hardly be remarked on. Anti-syphilitic treatment in very acute cases, unless commenced early, may be powerless to avert a fatal result. In fact, in the very acute cases of diffuse spinal meningitis following syphilis (which usually appears early, often in the first year after primary syphilis), presenting the clinical features of a transverse myelitis, an altogether gloomy view of the prognosis is taken by some authors, even granting that anti-syphilitic treatment be carried out.[4] A. Boettiger[5] says that in these very acute cases death generally results in the course of a few weeks or months in consequence of such complications as pyelonephritis, bed-sores, erysipelas, &c. Gilles de la Tourette[6] has apparently a more favourable opinion of the results of treatment, provided that it be commenced energetically and without delay.

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[1] Compare A. Boettiger "Beitrag zur Lehre von dem luetischen Rückenmarkskrankheiten," in *Archiv für Psychiatrie*, 1894, vol. xxvi., p. 690.

[2] See the abstract by G. Thibierge in *Annales de Derm. et Syph.*, 1891, vol. ii., p. 352.

[3] "Zur Kenntniss der Rückenmarkssyphilis," in *Deutsches Archiv für Klin. Med.*, 1889, vol. xliv., p. 246.

[4] Compare F. Scalfati, in *La Riforma Medica*, 1895, vol. i., p. 122.

[5] *Op. cit.*, p. 690.

[6] "Formes cliniques et traitement des myélites syphilitiques," in *Le Bulletin Médical*, Paris, June 10, 1896.

AN EXPERIMENTAL STUDY OF VISIONS.

BY MORTON PRINCE, M.D.

Physician for Diseases of the Nervous System, Boston City Hospital.

THE study of visions is of interest from many points of view; thus it would be interesting to understand the origin of the visions which have influenced the actions of certain historical characters, such as Joan of Arc and Catherine of Sienna. It is said that even Bismarck had a vision which he believed to be supernatural.

It ought to interest the theologian to understand the psychical origin of visions, like those of Luther, Savonarola, and hosts of saints, which have had such influence upon theological ideas. If it had been understood that such visions were only the pictorial representation in consciousness, according to natural psychical laws, of the fleeting thoughts, prayers, or beliefs perhaps long forgotten, with which the religious enthusiast had occupied his mind at one time or another, the influence of the Church might have been differently exerted.

For those interested in psychical research, a study of visions may tend to throw light on many seemingly "occult" phenomena; and for the psychologist, it may be possible to learn, from a study of the laws of the development of visions, other facts regarding the organisation of the mind; while, finally, by such a study, the alienist may be enabled to better understand the hallucinations of the insane. So that from many points of view the subject presents a field for interesting research.

As the nervous system acts by general laws, what is true of the origin and mode of development of vision is probably true of hallucinations of the other senses, of which those of hearing are the most important; so what we have learnt true of one, we can predicate as likely to be true of the other. Thus, a study of visions may also enable us to understand those hallucinations of hearing, which, like visions, have figured so conspicuously in the religious history of the past, and which play so large a part in the diseases of the mind.

It is well known that some people have the faculty of being able to create visions at will by intently gazing at an object. A crystal ball is commonly used for the purpose; the subject gazes into the crystal and, after a few moments of mental concentration, sees something therein—a scene, a person, or anything else, as the case may be. The phenomena have been called crystal visions from their mode of production. They are not particularly rare, and some, though not very many, experimental observations have been reported. In former times these visions played a part in occult lore, and, like modern clairvoyance, served to impose on the credulous. So that this part of psychology had its prototype in mysticism, just as chemistry may trace back its origin to alchemy, and astronomy to the romances of astrology. It is not so well known that it is the fashion for "spiritualistic mediums" to artificially cultivate the art of seeing visions, the method in use for the purpose being practically that of crystal gazing.[1] The hallucinations of hearing, which these same mediums profess to be communications with spirits of another world (and which some otherwise intelligent people believe), probably have an origin similar to that of visions, and are worthy of study from the point of view of this hypothesis. Visions created artificially by crystal gazing probably do not differ in their mode of genesis from those occurring spontaneously in normal people, and, there are good reasons for believing, may be the product of the same psychical laws as are some hallucinations, at least, of the insane. It may therefore be inferred without much danger of error, that a study of artificial visions will throw light upon the genesis of the spontaneous variety. The experimental method in favorable subjects allows us to study the phenomena with a fair degree of method, and to so alter the conditions as to vary the etiological factors.

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[1] A patient of mine having the curiosity to learn something about mediums, undertook (against my advice) to have herself developed by a professional. The "professor" claimed to have developed a very celebrated medium who has since been the object of study by Dr. Hodgson, Professor William James, Professor Lodge, and others. My patient told me that her education consisted in gazing intently, in conjunction with the professor, at a spot in the middle of a table, until she saw a vision at that spot. This was repeated at each lesson. If I remember rightly, a low diet and fatigue were coincident factors. My patient found she was successful beyond her courage, and becoming alarmed, reported to me. These visions, I think, soon got beyond her control and came against her will, but a cessation of the experiments and a little hygienic treatment stopped them.

It is to be hoped that further work will be done in this field. I have had an opportunity to make some experiments of this kind on a particularly favorable subject. In most of the observations reported hitherto, it has not been possible to thoroughly investigate the relation of the vision to antecedent events in the subject's life, beyond the evidence of the waking memory of that person. But in this instance, the fact that it was possible to hypnotise her and obtain *two additional and distinct personalities, three in all*, each with distinct memories, gave an opportunity to search in the hidden depths of consciousness, and obtain information about facts long forgotten by the normal personality. Further, it was possible to experimentally study the relation of the sub-conscious personality to, and the influence of this upon, the production of visions.

To understand this it is necessary to state a few facts regarding the subject. Miss X. suffers from what is ordinarily called neurasthenia, or hystero-neurasthenia, but what I think is more correctly called hysterical neurasthenia. But she has no physical stigmata, excepting a possible contraction of the field of vision.[1] She is easily hypnotised; first passing into a state which resembles that of the ordinary classical deep stage. After waking there is complete loss of memory of this state. During hypnosis she is very susceptible to suggestion; but on the whole there is nothing very remarkable about the mental condition, which does not materially differ from one of the deeper stages of hypnosis as ordinarily observed. The only points to which

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[1] At the time these observations were made this seemed to have disappeared, or, at least, could not be recognised by the finger test.

I would call attention are that Miss X. in this stage of hypnosis knows all about Miss X. awake, and apparently remembers all that Miss X. remembers and some other things besides, which she has forgotten when awake, but Miss X. when awake knows nothing of what has occurred in this hypnotic state. For convenience' sake, I have called Miss X., in this stage of hypnosis, X. 2, as distinguished from Miss X. or X. 1.

Now, on commanding X. 2 to sleep more deeply, there appears a third personality, whom I have called X. 3. This person knows all about both Miss X. and X. 2, everything they do and think, but is unknown to both. Her memory, also, in some respects, is much fuller than that of either Miss X. or X. 2, so it comes about that X. 3 can tell much in the past life of Miss X. that that person has forgotten, and can explain much that the waking personality is at a loss to account for. X. 3 even knows all about many of the little absent-minded and half-voluntary doings of Miss X., nor does she hesitate to voluntarily tell of them, although Miss X. is morbidly and unnecessarily reserved about her whole life.

I have been enabled to record a large number of interesting psychical phenomena which were exhibited by this patient, and which I shall report at another time, but the above facts are all that it is necessary to mention in this connection. It may only be added that the characters of the three personalities were very different and distinct. Miss X. is reserved, morbidly conscientious, self-contained, serious, differential, and dignified. X. 2 is sad, serious, and gives the impression of weariness and suffering. X. 3 is flippant, jovial, free from all physical infirmities, full of fun, reckless, and contemptuous of Miss X., whom she stigmatises as silly, stupid and dull, and in an apparently heartless way enjoys every trouble that comes to her. X. 3 always speaks of Miss X. as "She,"[1] and insists that they are different persons—that they don't think the same things or know the same things. She speaks of X. 2 as "Miss X.

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[1] In the following account, She, with a capital S, always refers to Miss X (X. 1).

asleep." This much will explain the conditions under which the following experiments were made. A study of the results obtained shows that the visions could be divided into three groups.

(*a*) Those which were revivals of past visual experiences, either conscious or unconscious.

(*b*) Those which were not revivals, but largely newly created visual representations, of a past experience other than visual.

(*c*) Those which were neither revivals nor representations of any past experience (visual or other) so far as was known.

Naturally some visions partook more or less of the characteristics of two or more groups, being partly revivals and partly new creations; nevertheless, all could be placed in one or other group according to the chief characteristic.

(*A*) *Experiments in which the visions were revivals of past visual experiences,* either conscious or sub-conscious.

Experiment 1.—In this and all of the experiments an incandescent electric light globe or lamp was used instead of the ordinary glass ball. Of course the bulb was not connected with the electric wires or circuit, but held free in the hand of the subject as if it were a mirror. This was the first experiment of this kind made with the subject, and in fact the first I had ever made. The idea of making the experiment only occurred to me at the moment, and I picked up the lamp which was lying on my desk without previously having given any intimation of the nature of the experiment to Miss X., who afterwards told me she had never heard of such phenomena. She was not, therefore, forewarned, and had no idea what was going to happen beyond that suggested by my first remark, which was that she should look into the glass globe, and she would see some thing in the past or future. Nothing specific as to what she should see was suggested. Miss X., after looking a few seconds, exhibited distinct evidences of fright, so that she kept taking her eyes off the bulb to avoid seeing, and obliged me to insist upon her looking. She afterwards explained this fright as due to a feeling of uncanniness. As she looked she described to me what she saw. She saw a young woman in a low-necked dress, blonde hair, about twenty-three, sitting in a chair near what was apparently a chimney-piece in a strange room. She was laughing. Standing in front of her was an older man with a dark beard tinged with grey. He was scolding her hard, but the young woman was laughing. Miss X.'s face during this time was expressive of very lively emotions, and I was obliged to calm her and assure her that it was all right, and there was nothing to be afraid of.

Here I may mention, once for all, the objective characteristics of these visions as they appeared to the subject and were described by Miss X., and also Miss X.'s mental condition at the time as I observed it. The visions were not seen like small objects reflected in or on the glass bulb, but Miss X. stated that the bulb disappeared, and she saw before her the scene she described, which appeared real, the characters being life-size and like living persons. I should infer that she saw them much as one sees the characters on the stage of a theatre, but she saw them, nevertheless, where the bulb was. Perhaps the illustration of looking through an opera-glass at the stage is apposite, for she several times at first broke off from her inspection and examined the bulb to see if there was any explanation of the vision to be found in the glass, much as one who had never seen an opera-glass or a kaleidoscope might examine it for this purpose. To me as I observed her she appeared like one who, at a theatre, was completely absorbed by the play, and in that sense was unconscious of surroundings, but not at all in a trance state. Her absorption and the exceeding mobility and expression of her face when describing a vision gave the impression that she was entirely oblivious of myself and surroundings, until spoken to, but not as one hypnotised, but rather as one who is intensely absorbed in the scene of a theatre and has forgotten where she is. Every feeling, timidity, surprise, interest, seemed to be expressed by the play of her features, and at times, especially at first, she seemed rather frightened by the uncanniness of what she saw. Most of her descriptions were given in answer to my questions, which were continuous but never suggestive. They were simply expressions like "What else?" "Go on," "Is there anything more?" &c. It may be noted, then, as psychologically of interest, that the visions appeared like ordinary hallucinations or vivid dreams, the scenes real, of life-size, but *dissociated* from her surroundings, and not as part of them.

To return, Miss X. said she could not see anything more. (It was not noted whether the vision cleared spontaneously.) Upon being interrogated she could give no explanation of the scene. She could not remember ever having seen the man, or the girl, or the place before. I insisted that the vision must have been a part of her past experience, but she could not recall anything like it to memory. I then hypnotised her and X. 2, the second personality, appeared. X. 2 also said she could not remember having seen either of the characters or the room before. She was then placed in deeper hypnosis, and the third personality, X. 3 appeared. X. 3 immediately, as soon as present, began to smile, then burst out laughing, and at once said she recollected the whole incident. It was at San Antonio, Texas, about seven years ago, when "She" was sixteen years old. They had left the dinner table, she said, and were going upstairs to their room, and on the way passed by an open door of a room. In the room were this young woman and the man, and he was scolding away, and she did not seem to mind it. They were strangers, and "She" only saw them once afterwards, driving in a carriage. X. 3 was highly amused at the recollection and the experiment. She was then awakened, and I told Miss X. (without giving the source of my information) that she had seen these people in San Antonio, Texas, and gave the particulars as above. She still failed to recollect the incident. The next day, however, she returned and said that after going home she had gone over in her mind her experiences in San Antonio, and by tracing successively in her mind the different events that had occurred there she had succeeded in bringing back into her memory the whole incident of the crystal vision. In answer to my objection that her recollection was an hallucination and was merely what had been suggested to her by me, she gave a great many more details preceding and following the event of the vision, showing that her memory now is much fuller than that which was shown in the vision. The scene in the room was a mere casual observation, and was not apparently of a nature to make an impression at the time, excepting that it produced some sort of unpleasant feeling, which she always feels when she hears people scolding. I noticed that she manifested some mental discomfort when she described the man in the vision scolding.

The conclusions to be drawn from this experiment are: First, that in this class of cases the hallucination represents a past visual experience—a visual picture was reproduced as a whole. Secondly, that the hallucination is in association with certain sub-conscious groups of mental states or personality (X. 3), but is not in association with all sub-conscious groups (X. 2). Whether it is more than in association with the group X. 3, that is an integral part of the sub-conscious group in the sense that it was X. 3's vision, the experiment does not allow us to determine. It is odd that of all the experiences of her past life such a trivial one should have been selected to emerge from out of the depths of subconscious life into consciousness. The continuous action and succession of events in this and other visions should be noted.

Experiment 2.—Miss X. looked into the globe and was highly amused. She saw Dr. C., first in a room of the Massachusetts Eye and Ear Infirmary, and then walking down the corridor. She told me that she had seen him there, but never doing what he was doing now. He was fidgeting about in the most nervous way, and walking down the corridor in a funny way. She insisted that although she remembered seeing him under the same circumstances as in the vision, she never saw him acting in that way.

Hypnotised.—X. 3 is full of fun to-day and rather rollicking (on this day experiments Nos. 3 and 6 were also made); told about the visions and enjoyed the thought of them hugely; spoke with great rapidity, rattling them off as if she were anxious to tell a good story and enjoyed the telling. She said that one day She was in the Infirmary, in the large operating room, with Dr. C. He was on one side of the table and She on the other. He fidgeted and put up one foot on the table and then the other, and moved first one arm and then the other in a nervous way. Miss X. copied everything he did, but not intentionally. Dr. C. became so nervous he could not stand it and ran out of the room. X. 3 laughed heartily at the recollection. This experiment is only interesting as showing how forgotten and trivial experiences may appear in visions.

Experiment 3.—This is an extremely interesting vision, but as it represents a somewhat personal matter, I give only the main facts. Miss X. saw two persons walking down the street. One of these, Y., crossed a field to some rocks by the sea shore, where Miss X. was sitting looking out to sea, with her back to the person approaching. Miss X. was about sixteen years of age, dressed as a young girl with her hair down her back. The approaching person went up behind her. She did not see him at first. He had a wild expression in his face, as if insane. "This," she declared to me, "is not true; he never looked like that." It was very disagreeable to see this expression on his face. She repeated "He looks insane." He took some letters or papers out of his pocket and tore them up, and crushed his watch, which he had in his hand. He acted wildly. Miss X. kept repeating with some fervour "This is not true," "This is not true." In explanation, she remembered that when she was a girl about sixteen the main incident happened; she did see this person walking across the fields to where she was, but she did not see him approach, and none of the rest was true. He simply came up behind her and spoke to her, &c. He did not do any of the things she saw him do in the vision.

Hypnosis.—X. 3 said it was all true. That She (Miss X.) saw him first walking in the street and across the fields, but that Y. came up close behind her without her seeing him. Then She saw him and spoke to him, but when She turned her face away he looked fierce, and crunched his teeth. He had his watch in his hand, and crushed it. X. 3 insisted that She did not see this, did not see the expression of his face, because every time that She turned round he smoothed his face out, and when She turned away her head this fierce expression came again. When I asked X. 3 how she could see that if her head was turned, she kept saying, "I did see it, but She didn't. She doesn't know. I saw it, and that he looked that way. She doesn't know he crunched his watch and tore the papers." (It would appear probable, assuming all this to be literally true, that Miss X. must have caught glimpses of this out of the corner of her eye, making use of peripheral as distinct from central vision. These glimpses were not distinct enough to make a conscious impression, but were seen by the hypnotic self, X. 3. She may have heard the crunching of the watch and of the papers. It is more than probable that the details in the crystal vision are largely exaggerated, being created unconsciously out of the inferences which Miss X. or X. 3 drew from what she saw, a peculiarity of these visions, which is illustrated by some of the other experiments. X. 3 probably didn't see as much as she thinks she did, but womanlike, gives her inferences instead of her actual observation.

Experiment 4.—X. 3 now (after explaining the last vision) volunteered the following remarkable story, telling it with great gusto and as a joke on Miss X., and speaking with great rapidity, so that it was difficult to follow the sequence of events. The language was substantially that of X. 3, but condensed. "She" yesterday received a letter from a photographer. She had it in her hand while walking down Washington Street, and then put it into her pocket (side pocket of jacket) where She kept her watch and money (bank notes). Then as She walked along She took out the money and tore it to pieces, thinking it was the letter from the photographer. She threw the money into the street. As She tore up the money She said to herself "I wish they would not write on this bank-note paper." At my request X. 3 repeated the words of the photographer's note, which was to say some photographs were ready. As to the money, there were twenty dollars, two ten dollar notes; this, at my demand X. 3 counted mentally, with some difficulty and concentration of thought. X. 3 manifested considerable unwillingness to show me the letter, which she said was in "her" pocket still, and which She still thought to be money. Finally, after some insistance, she did so. It was folded up into a small square, just as one often folds bank notes. The language of the photographer's note was identically the same as quoted by X. 3. X. 3 said that "She" was absent-minded, and thinking of something else, when She tore up the money. I then gave X. 3 the note to put back in her pocket preparatory to waking her up. This impish, hypnotic personality remarked upon what a joke it would be when She found it there instead of the bank notes. I will say here the heartless, cold-blooded delight which this almost Iago-like personality found in the loss of the money, which was a very great one to Miss X., was appalling. To X. 3 it was a splendid practical joke.

Miss X. was now awakened. I asked her whether she did not have some money and had not received a letter from a photographer. She said "Yes," but seemed to think it rather queer my asking these questions, but by this time she had become accustomed to being astonished. In reply to a series of questions she said she had not the letter with her, but had torn it up and thrown it into the street (Washington Street). The bank bills she had in her pocket. They were two ten dollar bills. I asked her to show them to me. She put her hand in her pocket to take them out and brought out the photographer's letter. She evidently received a shock although she tried not to show it. I asked her where the bills were, and after searching her pocket she insisted that she must have left them at home. I remarked that she must have destroyed them by mistake instead of the letter; she refused to admit it. I pointed out the circumstantial evidence; she recognised that it was suspicious but could and would not believe it. The loss meant a great deal to her, and she evidently encouraged herself with a forlorn hope. I then said, taking the glass globe, "We will see whether it is not true. Look in and you will see what you have done." At first she saw only indifferent things; then I said, "Think of bank notes and the feeling of tearing them up." Now, to her astonishment, she saw herself walking along Washington Street and putting the letter in her pocket, then taking out what looked like bank notes, that is green pieces of paper, and tearing them into pieces and throwing them into the street. The vision in all its details corresponded to the account given by the hypnotic personality. Miss X., in answer to my question, said she had once before done a similar thing, namely, in a state of absent-mindedness, she had thrown some bank notes into the fire and had taken the letter in which they were "down town" to pay some bills. The next day Miss X. reported that she was unable to find the money at home, and was satisfied the vision was true. In hypnosis, X. 3 now volunteered the information that Miss X. ("She") was so much upset by the loss of her money, that in the middle of the night She "had to get up" in her sleep, without knowing it, and had taken the remainder of her money and hidden it under "that floppy thing" on the table. "It was now under a red book, a blue book, and that floppy thing" (by this is meant either a table cloth or a folded up piece of material). "She" knows nothing about it, and thinks She has lost the money and has none left. X. 3 does not know how much money there is. She is very reticent, does not like my knowing, and will not admit the fact. (To have this understood it would be necessary to go into a rather lengthy explanation of the circumstances of Miss X.'s life, which I hardly feel called upon to do. I will merely say that Miss X. had come to Boston to be under my professional care and was in lodgings and had a certain fixed sum for her expenses; the loss of this money meant financial bankruptcy.) Without further discussion, and *without disclosing my knowledge*, I then presented a glass globe to her; told her to think of the money and she would see what had become of it. She looked into the globe and saw herself in bed in her room. She then saw herself get up, her eyes being closed, and walk up and down the room; she then saw herself going to the bureau drawer, taking out her money, going to the table, taking up the table cloth with the books, putting the money on the table, covering it with the cloth and putting the red book and the green book on the top of it. The vision thus exactly corresponded[1] to the statement of X. 3. Miss X. reported at the next visit that she had found the money where she had seen it in the globe.

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[1] Excepting the colour of the book, "blue" or "green." This escaped my attention at the time; perhaps it was blue-green, and looked blue to X.3 and green to X., or it may be an error on my part.

Remarks.

It is noteworthy that in this experiment, as well as the preceding one, the vision was a representation of the hypnotic or subliminal consciousness, and not of the waking consciousness. In experiment No. 3, certain things were portrayed in the vision which the waking consciousness (apparently) never perceived, while in experiment No. 4 (first part, destruction of bank bills) an absent-minded act was represented, and in the second part a somnambulistic act. It might be held that the first vision of the bank note episode might have been created out of my statement to her, if it were not for the minute fidelity with which the vision corresponded with the account given by X. 3, and the corroborating evidence of the second vision when I gave no hint of what would be seen.

(*B*) *Experiments in which the hallucination was not a revival of a past visual experience, but was largely a newly created visual representation of a past experience other than visual.*

Experiment 5.—I will give this in the brief language of my notes: Miss X. looked again into the globe; she saw a room with a bed in it. There was a figure in the bed; the figure threw off the bed clothes and got up. Miss X. exclaimed "Why, it is I." (Appeared rather frightened at what she saw, but went on to describe it, largely in answer to my promptings, such as "Go on," "What do you see?" &c.) She saw herself walking to and fro, up and down the room. Then she climbed on to the window sill which is the deep embrasure of a mansard roof. Then she climbed outside the window and from the sill looked down into the street. It was night—the street lamps were lighted, there was also the gas light in the room. As she looked down she felt dizzy. Here Miss X. turned away frightened, saying she felt the same dizziness as if she were standing there. She soon continued. She saw herself throw an inkstand into the street below which she had seen herself take before climbing on to the window. Miss X. was again obliged to stop looking because of dizziness. After a time she returned to the globe. She saw herself go back into the room and walk up and down; the door opened and she jumped into bed and lay quiet. Miss L. (a friend) entered, went out and returned several times;

brought a poultice which she put on Miss X.'s chest; Miss X. herself remaining quiet. Then Miss L. went out and Miss X. got up and took the poultice, rolled it up into a little bunch and hid it in a corner, putting a towel over it. Here the experiment ended.

Miss X. stated, on being questioned, that she could not remember any incident like the vision, excepting that she recognised the room as the first one she occupied when she came to Boston four or five years ago. It was in the top story of a house on Columbus Avenue; she was ill there, and Miss L. took care of her. But she did not remember ever having climbed on to the window, or having thrown an inkstand, or any of the incidents of the vision. She could throw no light on the affair. She was now lightly hypnotised and X. 2 was present. X. 2 could add very little to Miss X.'s statement. (My notes of X. 2's memory are somewhat confused; it is not plain from them whether or not she remembered ever having done any of the things seen in the vision, although it is stated that she had an imperfect memory of the incident. It is possible X. 2's statement as noted was mixed up with that of X. 3, but X. 2 remembered being ill in the room described, and that ink was found in Miss X.'s shoes; X. 2 did not know how it came there, but Miss L. had said that Miss X. had poured ink in her shoe. At any rate X. 2 could not explain the incident as X. 3 did later.)

Deep hypnosis; X. 3 appeared. With great vivacity and amusement X. 3 explained the whole scene. (As was customary with her, X. 3 spoke of Miss X. (X.) as "She," as if it were an entirely distinct person and not herself.) "She" had pneumonia and was delirious, and She imagined She was on the sea shore and was walking up and down the sand. This was why She walked up and down the room, and She stuck her toes in the carpet thinking it was the sand. There were rocks there, and the window sill was one of them, and when She climbed out upon the window sill She thought She was climbing upon a rock, and She took up a stone, as She thought, and threw it into the sea. This was the inkstand that She threw into the street. Then when she took the poultice and hid it in the corner She thought She had buried it in the sand. She had not poured ink into her shoes, but her hand shook and She had spilled it into her shoes. Miss L. seeing the ink stains had inferred that Miss X. had poured the ink into the shoes, and had told Miss X. so. Hence the statements of X. and X. 2. X. 3 was highly amused at all the mistakes of Miss X.'s delirium.

Remarks.

It is obvious that this vision was not a revival of an optical experience of the past, for Miss X. could not have seen herself as she did in the vision. The hallucination was rather a new creation corresponding to, and illustrative of a memory of, a past event. But into this new creation a certain number of past optical images entered, as in the scene in the street and the room. Yet even these were originally seen by the delirious consciousness in a different way, that is as if it were the sea shore. The vision rather corresponded to the way in which the third personality, X. 3, would have seen delirious X. 1 and her surroundings if she could have seen them. This suggests the relation of the hypnotic personality, X. 3, to the delirious consciousness. X. 3 observed the delirious Miss X., and was cognisant of her thoughts just as she was of those of the normal Miss X. Just as X. 3 knew all about X. 1 when in health, so she knew all about X. 1 when delirious. But normal X. 1 had no recollection of delirious X. 1. The hypnotic consciousness is not the delirious consciousness, but bears the same relation to it that it does to the normal consciousness. X. 3 was normal while X. 1 was delirious. It is a startling idea, that of there being beneath the delirium of a person in fever another healthy personality, a mind unchanged, which is observing everything done, thought and said by the delirious person. It is worth noting in passing that when Miss X. saw herself looking into the street, she at that moment actually felt dizzy. That is, as the scene appeared real to her, looking down into the street of the vision made her dizzy.

Experiment 6.—This experiment was interesting, as it showed how a vision could be constructed out of certain past familiar images and certain other experiences of which no optical images could have been received; a new synthesis of images being created by the force, if I may use the word, of imagination and known facts. Looking into the globe, Miss X. was astonished to see herself sitting on a sofa, the identical sofa on which she was then sitting. *Her eyes were closed*, and she was smoking cigarettes. While Miss X. was looking at herself in the globe she several times turned to look at the sofa on which she was sitting to compare it with that in the globe. They were the same. Her whole expression was one of extreme astonishment and chagrin. She indignantly repudiated the fact, said it was not true, she was was not in the habit of smoking cigarettes, and had never smoked one. In giving a further description of herself in the vision she said her eyes were closed and she looked foolish. The explanation of this vision was that the day before, as a practical joke, I had given a cigarette to X. 3, who had taken it in a spirit of fun and smoked it clumsily, showing plainly she had not been accustomed to smoking. X. 3 laughed and made merry over the prank, saying it was a great joke on Miss X., who would be "awfully shocked" if She knew it. Miss X. pretended it was very wicked—as it was for her. The visionary Miss X.'s eyes were closed, for I had always obliged X. 3 to keep her eyes closed for reasons not necessary to go into here.

Remarks.

It is noteworthy that the vision, so far as it concerned cigarette smoking, represented an experience which belonged only to the hypnotic personality of which Miss X., the superior consciousness, never had any knowledge. From this it might be inferred that visions might represent absent-minded acts as in the case of experiment No. 4. Secondly, the contents of the vision were partly previous optical images received by the primary consciousness, that of Miss X., viz., images of the sofa and portions of Miss X. (X. 3 had never had her eyes open), and partly were syntheses of the third personality's sole *knowledge;* thus the vision was made up of the experiences of two personalities, and only in part of past images. As she had never *seen* herself smoking, the vision was largely an optical representation (not a revival) of impressions from other senses.

(*C*) *Experiments in which the vision did not reproduce or represent any past experience, visual or other, so far as was known.*

Experiment 7.—Miss X. looked into the glass globe. She saw an old lady standing in a very steep street leading up over stones; there were steep rocks on one side. The street was like nothing she had ever seen before in this country. (She said *this country*, as if to emphasise the fact that it looked foreign.) She had never seen the old lady before. As she looked, a man on horseback appeared, and the horse knocked the old lady down. The latter seemed to be hurt. Here Miss X. turned her head away as if alarmed. On another day Miss X. again saw the same vision. The man's features were also strange to her. The most minute inquiry failed to elicit any fact that would throw light on this vision. Miss X. said she had never been abroad, and had never seen any place resembling that of the vision, nor knew of any such accident. When hypnotised neither of the other two personalities (X. 2 and X. 3) had any recollection of any such place or event. I am disposed, therefore, to believe that it was probably a visual representation of something she had read, or of some picture she had seen, and which would readily pass out of the recollection of even the hypnotic self. As she described the street in detail it recalled places to me I had seen in Europe, or pictures of foreign scenes.

Experiment 8.—Miss X. had lost a scarf pin she valued. I suggested that she should try to find it by looking in the globe. After looking a few minutes, she saw a bed-room with a brass bedstead, and on the dressing-table was a pin-cushion (described in detail), and in the pin-cushion were several pins. Amongst them she recognised her own, and exclaimed somewhat excitedly "Why, that is my pin!" Involuntarily she reached out to seize it. During this her face was very expressive, and showed great astonishment. She did not recognise the room, and was certain she had never seen it before, but thought it a city room and not a country one from its general appearance and furnishing.

In hypnosis both personalities were also ignorant of this room, but X. 3, being asked to tell the last time that Miss X. had the scarf pin, recollected perfectly. "She" (Miss X.) came into her room (in the country, not in Boston) one day with a very severe headache, took the pin out of her dress, and stuck it into the head of the couch, then doubled up the pillow and placed it over it, and lay down on the couch with her head on the pillow and rested. "She" forgot all about the pin, and had no recollection of putting the pin where She did, and, in fact, did it rather absent-mindedly. This was several months ago, and X. 3 presumed the pin was gone. On Miss X. being awakened, I told her what she had done with the pin, but still she had no recollection of the affair.

Remarks.

Probably in this case the vision did not correspond to any known fact in Miss X.'s experience. The room, I suppose, might be some room she had casually seen but forgotten, but she could not very well have seen her pin in it. Perhaps she might have thought, at some time, that the pin had been found or taken, and was in the possession of someone. Thus the vision might have been constructed in much the same way as the cigarette one (experiment No. 6). I was unable to learn whether X. 3's story was correct or not, but it probably was, as I had always found her positive recollections accurate. This experiment also illustrates, like No. 4, the effect of suggestion in determining the character of the vision; that is, a vision of a particular thing or subject in preference to any other vision. I told Miss X. she was to look into the globe for information of the pin, when she saw the contents of X. 3's consciousness as a vision; in other words, it was not her own subconscious personality that came up from below into consciousness as a vision, on the contrary, it was X. 3's proper consciousness. This seems also to have been the case in experiments Nos. 3 and 6.

I wish that further observations had been made on this subject, especially with a view to determining how far the contents of a vision could be shaped by suggestion, and the relation of visions to beliefs, and other thoughts; but various considerations made it desirable to interrupt the experiments at this point. As the laws governing one sense are probably the same as those for another, it is likely that the antecedent relations of hallucinations of hearing are the same as those of hallucinations of vision. This inference is somewhat speculative, but it gives additional value to a knowledge of the origin of visions. If it be true, as is most highly probable, that visions may be an optical representation of pure thoughts and beliefs, this fact gives us something with which to answer Griesinger's question, "Why does the insane patient believe in his hallucinations?" It would seem as Hirst[1] well says, "The patient does not believe himself to be the son of the emperor, because the voice told him so, but he heard the voice making this statement because he believed it."

CONCLUSIONS.

(1) Visions in sane persons may be revivals of past visual experiences, which originally may have been conscious or sub-conscious. The original sub-conscious experience may have occurred in a moment of absent-mindedness, or may not have been sufficiently intense to have entered consciousness, or (rarely), may have occurred in somnambulism.

(2) The vision instead of being a revival, may be a newly-created pictorial representation of a past experience other than visual. That is to say, past impressions of one or more senses (touch, hearing) and actions may translate themselves into a representation by another sense as a vision.

(3) It is probable, though not proved, that a vision may not reproduce or represent any past experience, visual or other, but may be newly created out of something the subject has read, heard, or thought. The inference from, and passing thoughts about, known facts may weave themselves into visions. This was probably the origin of the visions of Joan of Arc, and religious enthusiasts.

(4) Visions may partake more or less of the characteristics of these classes, being partly revivals, partly representations of actual non-visual experiences, and also of the subject's knowledge, inferences and thoughts.

(5) Generalising, it is possible that hallucinations of the other senses, especially of hearing and as exhibited by trance mediums, may have a similar origin and composition.

(6) Analogous phenomena may be observed in the attacks of hysterics where the passing thoughts in the normal state may appear as insistent ideas in the attack.

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[1] "The Psychical Mechanism of Delusions." The *Journal of Nervous and Mental Diseases*, March, 1898.

(7) It is probable that thoughts which have strongly absorbed the mind and expressed the longed-for ambition, or the ideas and beliefs of the subject, may appear as visions. The subjects then are apt to look upon them as inspirations. In this way have arisen the visions of political personages like Joan of Arc, Bismarck, and religious enthusiasts like Luther, Fra Angelico, Catherine of Sienna and others.

(8) Visions, artificially created, may be representations and revivals of the experience of the hypnotic personality, of which experience the waking consciousness has never had knowledge.

(9) Impressions on the sense organs which never entered consciousness (therefore neither known nor remembered), may afterwards appear as visions.

A CONTRIBUTION TOWARDS THE DETERMINATION OF THE ENERGY DEVELOPED BY A NERVE CENTRE.[1]

BY VICTOR HORSLEY, F.R.S., F.R.C.S.

I HAVE first to return my very sincere thanks to the Society for the honour that they have conferred upon the surgical side of Neurology and upon myself in electing me to their presidential chair, and I appreciate the honour the more since it is a position which has been occupied successively by almost all my teachers, prominently among whom is my immediate predecessor, Dr. Savage.

It is the established custom for the person just inducted into the chair to deliver an address, and I am very glad to embrace this opportunity of communicating, if in a somewhat preliminary manner, the results of an investigation which I planned some years ago, and upon which I have also worked from time to time partly in collaboration with Dr. Veraguth and Dr. Christiansen.

The subject matter of this research is an attempt to estimate quantitatively the "work"[2] done by the central mechanisms of the central nervous system when these discharge their nerve energy; for example, to discover, if possible, the amount of energy developed by the centres in the cortex cerebri and in the spinal cord respectively, the relative volume of the two mechanisms being so very different, see fig. 1. Apart from the general scientific interest of prosecuting this study of nerve energy for its own sake, this subject I venture to think has a considerable importance in relation to the epileptic convulsion, and therefore I bring it forward to-night as a matter of concern to both the physiologist and the clinician.

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[1] Presidential Address to the Neurological Society.

[2] Of course the word "work" is not used here in its scientific sense, since we do not know the nature of nerve energy or nerve force. The words "energy" and "force" are also to be accepted in their popular meaning.

When we speak of the discharge of nerve energy from a nerve centre it is not a little disturbing to find that we know nothing about nerve energy except the rate of its transmission along the nerve fibres and the circumstances under which it is evolved. It has of course been compared to all forms of so-called physical energy, and Newton himself speaks of a nerve impulse as amounting to a vibratory disturbance of the particles of the nervous system.

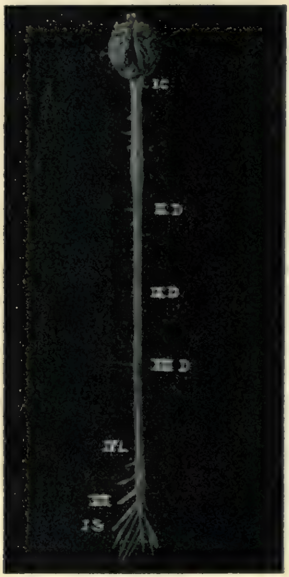

Fig. 1.

Central Nervous System of the Cat.

Nerve energy cannot therefore be measured with any apparatus known to us at present, and therefore if we are to attempt to study its development quantitatively the only available method of doing this is by indirect means such as quantitative measurement of either some constant correlative phenomenon or of any physical effect directly produced by its activity. I would suggest that there are two possible ways of arriving at some sort of conclusion: these are (1) electrical, (2) mechanical.

(1) *Electrical Method.*—Of these the first is the measurement of the "negative variation" of Du Bois Reymond or "action current" of Hermann, which accompanies, of course, every single so-called nerve discharge or impulse, and which, in a summated form, may be accurately enough estimated by the galvanometer or electrometer.

It will be realised that the electrical method of examining the degree of discharge of a nerve centre is only the quantitative observation of an electrical change in the nerve channels running from the nerve centre which is the seat of stimulation; in other words, the electrical method offers of course nothing absolute but provides the determination of a very important parallel phenomenon, such phenomenon being developed under such circumstances as make it at least likely that a quantitative relationship exists between it and the discharge of nerve energy. So that in a measure the degree of electrical change may be said to be an expression of the degree of development of nerve energy. The proof of this was given by Professor Gotch and myself six years ago when we published in full[1] the results obtained by our method of investigating the functional activity of various parts of the nervous system. Among our facts we included a table giving the readings of the deflections observed when the galvanometer was connected with the spinal cord and the cortex cerebri excited and when, in addition, the general epileptic condition of the upper half of the body was simultaneously noted. I repeat here a table (constructed from experiments on four different animals) which shows clearly that the electrical change or summated action currents in the spinal cord was quite proportional to the degree of discharge from the cortical nerve centres.

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[1] *Phil. Transactions*, vol. clxxxii., p. 331, 1891.

Galvanometric Effects Produced in Spinal Cord of Cat by Cortical Excitation. (*Phil. Trans.*, 1891).

	Intensity of Excitation	Duration and Place of Excitation.	Galvanometric Effect	Muscular Movement
Cat (31) ..	8,000	5" left cortex	63	Slight fit
	8,000	,, right ,,	115	,, ,,
	9,000	,, left ,,	108	,, ,,
	9,000	,, right ,,	230	Good fit
,, (317) ..	8,000	,, left ,,	50	Very slight fit
	10,000	,, ,, ,,	162	Fair fit
	12,000	,, ,, ,,	235	Good fit
	8,000	,, right ,,	130	Slight fit
	10,000	,, ,, ,,	185	Fair fit
	12,000	,, ,, ,,	230	Good fit
,, (319) ..	10,000	,, left ,,	90	Slight fit
	12,000	,, ,, ,,	170	Fair fit
	10,000	,, right ,,	150	,, ,,
	12,000	,, ,, ,,	260	Good fit
,, (324) ..	6,000	,, left ,,	260	Slight fit
	8,000	,, ,, ,,	340	Fair fit
	10,000	,, ,, ,,	510	Powerful fit
		Average.. ..	17 = 3288	
			193	

I intend to pursue this branch of the subject with the advantage of the use of the capillary electrometer as recently perfected by Mr. Burch, seeing that with the electrical units now in use the disturbance of energy in the spinal cord channels can be expressed scientifically in terms of work and consequently be directly compared with the records obtained from the muscle by the mechanical method *vide infra.* This has been rendered possible by the recent determination by Gotch and Burch, of the true value of the single "negative variation" or "action current." The unsatisfied condition, however, of course, always still remaining to mar this method, namely, that we cannot know the *precise* scale of parallelism between the amount of nerve energy and the amount of the electrical effect, until some one discovers a means of directly recognising and measuring nerve energy.

(2) *Mechanical Method.*—By this expression I wish to convey the fact that advantage is taken of the connection

of certain muscles with certain nerve centres to accept the work done by the contraction of such muscles as representing the degree of nerve energy evolved from the centre the activity of which produces the contraction. For this purpose we require measurement of the "lift," *i.e.*, height and weight lifted, effected by the discharge of any given centre, as well as the total work done in a given time. I shall on this occasion speak of the "lift" accomplished by the muscle and not the areas of the curves obtained in the experiments which would give the total work done. Provided that the centre examined and motor nerve leading therefrom are subjected to a maximal excitation (maximal because since each of these experiments is a comparison and at the same time involves the excitation of structures very different in nature and excitability as nerve centres and nerve channels respectively are, naturally the stimulus must be maximal) then we know that we have reached the limit of mechanical effect obtainable from the part excited, and it then becomes possible for us to compare the effect obtainable from the muscle when directly excited from its nerve with that obtained as the effect of the discharge of nerve energy from the centre. From the comparison of these results we gain a relative value. Whereas in the case of the electrical method there is the drawback, as I pointed out, of want of a precise knowledge of parallelism between the correlated phenomena of nerve discharge or electrical "action current"; in this mechanical method we have a larger and more important source of complication and possible error which must now be noted. If we are

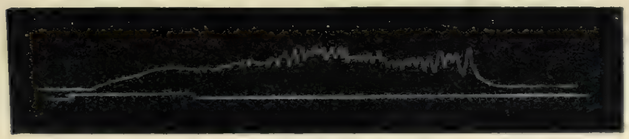

FIG. 2.

Graphic record of Muscle Contraction resulting from Cortical Excitation. (*Phil. Trans.*, 1891.)

applying the method to obtain an estimate of the activity of the centres in the cortex cerebri, it is a fair assumption that the discharge observed arises in the cortex cerebri, but there remains the exceedingly difficult question as to how far the bulbo spinal centres are, or are not, taking part and producing a certain share of the whole effect. To take a concrete example the question is, given excitation of the centres in the cortex we have to enquire whether the total amount of work done by the contracting muscle is due to the energy of the cortex, or due to the energy of the cortex plus that of the spinal cord; or, to put the matter yet in another way, the question is whether the spinal cord, under these circumstances, is acting as a mere conductor, or whether it is acting as a contributing agent. As regards the *character* of the contractions of the muscle, *i.e.*, tonic and clonic (see fig. 2), this problem was first satisfactorily approached by Franck and Pitres, who, in 1877, found

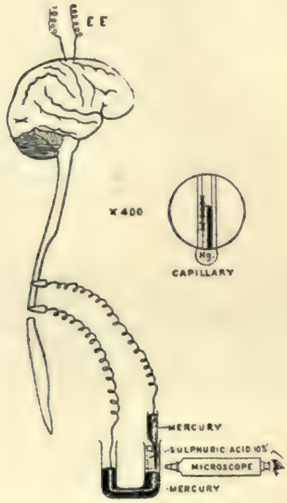

FIG. 3.

Schematic representation of electrometric arrangement for estimation of the descending changes in the cord.

by the graphic method that excision of the cortex and excitation of the subjacent corona radiata fibres was followed by practical abolition of the clonic element, so characteristic a feature in the effect derived from stimulation of the cortex. This result was confirmed by Bubnoff and Heidenhain (1881), Schafer and myself (1885). From this fact the conclusion was drawn by some observers that the tonic element was due to the lower (bulbo spinal centres). On this point a controversy arose between Ziehen[1] and Unverricht,[2] which had the effect of emphasising the essentially clonic nature of the discharge of the cortex centres. The only proof, however, that could be accepted as really bearing directly on the character of the elements of the cortical discharge was afforded by the electrical method of Gotch and myself,[3] where, by interpolation between the cortical and bulbo spinal centres respectively (see fig. 3), the fact that the cortex discharge consists of *both* tonus and clonus elements was finally established. (See fig. 4 and fig. 5, in which latter the action currents of the clonic spasms are

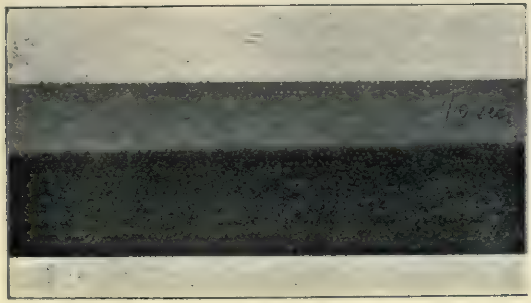

FIG. 4.

Reduced photographic record of the electrometric variations, the cord being connected to the electrometer and the cortex excited.

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[1] Chief paper "Zur Physiologie der Infra-corticalen Ganglien, &c." *Archiv f. Psychiatrie und Nervenkrankheiten*, B. xxi., p. 863, 1890.

[2] Chief paper "Ueber Tonische und Clonische Muskelkrampfe." *Deutsches Archiv fur Klinische Medicin.* B. xlvi., p. 418, 1890.

[3] *Loc. cit. supra.*

more highly magnified.) Still there remains the problem, how far in the intact nervous system does the spinal centre add to the work effected, since it is now clear that the *character* of the whole effect is entirely cortical. At present our data are imperfect and must remain so until by the electrical method the work done in the nerve channel between the cortical and the bulbo spinal system respectively has been measured. Now, as regards the mechanical method, I am going to suggest that for the purposes of the present subject this question of the source of the tonus contraction does not stand in our way of advancing the subject for the reason that it is possible to first obtain a maximal reflex discharge from the spinal centres, and by that means to discover that the cortex cerebri gives a discharge of more energy than that of the cord, or at any rate, during the first few seconds of the stimulation time. In fact, a relative estimation is easy to make when the effect of a cortical excitation is, as a matter of fact, so much larger than that derived from a direct stimulation of the isolated of the spinal cord. Certain further precautions have to be dwelt upon before we can expect this method, which I only put forward as a means

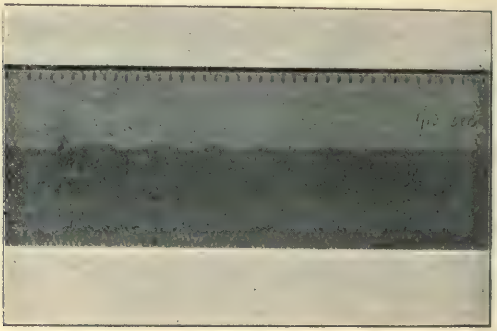

FIG. 5.

The clonic stage in fig. 4 recorded under higher magnification and more rapid rate.

of indicating the relative powers of the different nerve centres to fulfil all we hope from it.

Description of the Mechanical Method.

The mechanical method I have adopted in these experiments is as follows: All the initial operations were performed under ether narcosis. The cortex cerebri was exposed and kept warm. The muscles chosen have been the biceps brachii or the rectus femoris. The latter appears to be the more suitable muscle, certainly it does not become fatigued so soon, or, rather, the exposure of its nerve is not followed by so much shock as in the case of the biceps. An inextensible thread was carried from the muscle over a small pulley and attached to a small metal bucket running on steel guides and carrying a light aluminium marker. The total weight to be raised by the muscle was made equal to 100 grammes by putting shot in the bucket. This, together with three electrical signals, two for recording the moment of stimulation from the two separate induction coils, and the third, a seconds of time marker, was arranged to write simultaneously either on the paper of a Hürthle Kymographion or on the glass plate of Professor F. J. Smith's chronograph, in which the carrier was drawn forward by slow clockwork. Both sciatic nerves were exposed and ligatures passed beneath them, as also the right anterior crural nerve, where it is buried in the substance of the psoas muscle. A slip of indiarubber sheeting was passed under the latter, and fine platinum electrodes were hooked on the nerve in question lying between the folds of the india-rubber. By use of the second coil it was possible to arrange simultaneous excitation of the different structures. After a tracing had been obtained of the muscular spasm resulting from excitation of the cortex, then where possible a tracing was taken consequent upon excitation of the corona radiata, and when the observations upon the cerebrum had been completed, the spinal cord was divided at about the level of the sixth dorsal vertebra, and everything prepared for direct observations of the reflex movements. These were investigated in the following way:—both sciatic nerves were firmly tied with ligatures and divided peripherally. At the moment of ligature and of subsequent division a tracing was taken. The reflexes were ascertained by first exciting the central end of the left sciatic nerve, the right rectus femoris being always chosen; and if there was any effect obtainable, the direct reflex was determined by excitation of the right sciatic nerve (central end).

Relationship of the Different Nerve Centres.

I have not in the following research attempted to ascertain any influence of the cerebellum whether exerted upon the cortical or bulbospinal centres respectively. Its supposed function as a controller of the cerebrum, surmised theoretically by Gowers and established experimentally by Risien Russell is paralleled by Löwenthal's discovery[1] of its controlling influence on a certain (extension) share of the reciprocal innervation phenomena of the cervical bulbospinal centres. So, too, I have ignored the influence of the basal ganglia, because we do not know what their function is, and the idea that they participate in so-called purposive acts which we now know to be of cortical origin has, I believe, no real foundation in any scientific fact.[2] Moreover, it is not easy to see how they could experimentally be excluded by resection because of the close proximity of the fibres of the internal capsule passing from the cortex down to the spinal cord. The connection between the centres in the cortex and the spinal cord is, of course, of importance in the present question. Topographically it has been shown by the simple excitation method originated by Hitzig and Fritsch and so greatly extended by Ferrier, that the fronto-parietal region is alone in direct connection with the centres in the spinal cord, whereas the special sense areas, *e.g.*, occipital, temporal, &c., are in undoubted connection with some of the nuclei of the cranial nerves at the base of the brain and bulb. A further proof of this position was also obtained by Gotch and myself[1] by the electrical method, when we found by leading off different parts of the spinal cord to the galvanometer that excitation of other parts of the cortex, *e.g.*, of the occipital lobe, failed to evoke any electrical change in the spinal cord. The nerve centres therefore, which I have chosen for analysis by the mechanical method are those only of the limb areas of representation in the parietal cortex and the spinal centres for the limbs in the spinal cord.

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[1] Löwenthal & Horsley, *Proc. Roy. Soc.*, 1897.

[2] See Beevor & Horsley, *Phil. Trans.*, 1890, p. 49.

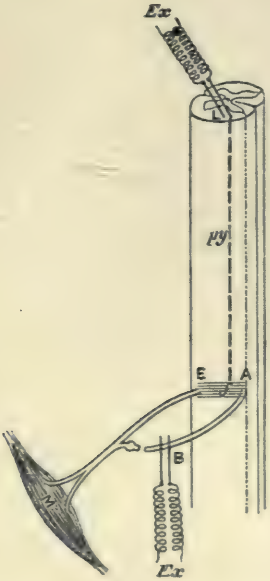

FIG. 6.

Diagrammatic representation of the reflex arc and connection with the pyramidal fibres (*Phil. Trans.*, 1891.)

Narrowing our considerations then to this degree we enter upon the next preliminary question. What is the nature of the direct connection between the cortical centre and the spinal cord centre looking upon the latter as an intermediate stage between the cortical centre and the muscle.

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[1] *Loc. cit. supra.*

The degeneration method has shown that the fibres of the pyramidal system descend from the cortical centres, pass into the spinal centres for the muscles, and therein come in an unknown way, into relationship with the large ganglion cells of the anterior cornua, from which extend an axis cylinder process down the so-called motor root and mixed nerve to the muscle.

By the electrical method Gotch and myself showed that there is a remarkable diminution in the electrical effect obtained from the spinal centres along the large nerve trunks to the muscles as compared with the effect obtained on the spinal cord by excitation of either the cortex or pyramidal fibres of the corona radiata, suggestive of the view that the spinal centre forms an intermediate station of considerable influence (see fig. 6.)

I have thus far dealt with this matter of the connection of the cortical centres and the spinal centres, because it is one of great importance, and because it enters into some considerations later as well, but it does not have a direct bearing upon the immediate *value* of the mechanical method since we find that just as is shown by the electrical method in the spinal cord, so the contraction of a muscle *i.e.*, work done by the muscle, varies directly with the amount of discharge from the nerve centre. As a matter, therefore, of relative proportion the activity of the muscle is undoubtedly a measure of the activity of the nerve centre. This being so we are then able to use the muscle to measure the relative values of the discharge of the different parts of the system. Before taking up the actual result obtained I would like to point out that by the electrical method Gotch and myself found that excitation of the corona radiata evoked an electrical change in the cord, which was slightly more than half that evolved when the cortex cerebri was excited. In this connection I must repeat that there is properly speaking no second stage or clonic after effect when the corona radiata is excited, and that the galvanometric observations are those of summated discharge effects.

So, too, the electrical effect in the nerve when the spinal centres are caused to discharge by reflex excitation give rather more than one seventh of the deflection observed when the cortex is excited. That the pyramidal system of fibres have practically the same degree of connection with the elements of spinal centres, as the fibres of the other channels is shown by the fact that the amount of the galvanometrical deflection obtained from the nerve when the cortex was excited was equal to that noted in the nerve when the discharge of the reflex spinal centre was passing down it.

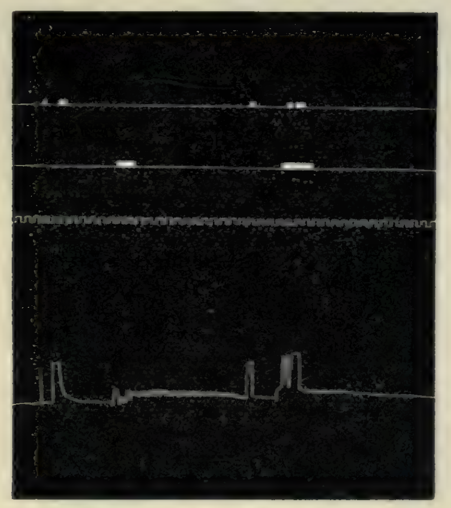

FIG. 7.

Arrangement of the Nerve Centres according to the Degree of Work Done.

In employing as the scale of comparison between the respective heights of the contraction of a muscle in raising the weight of 100 grammes I should state at once that I have never seen any discharge from the nerve centre to result in a contraction of the muscle exceed in height that obtained by stimulating the nerve to the muscle, and in fact no nerve centre by any means that I have employed and described is capable of evoking as much work from the muscle as excitation of the motor nerve to the muscle produces.

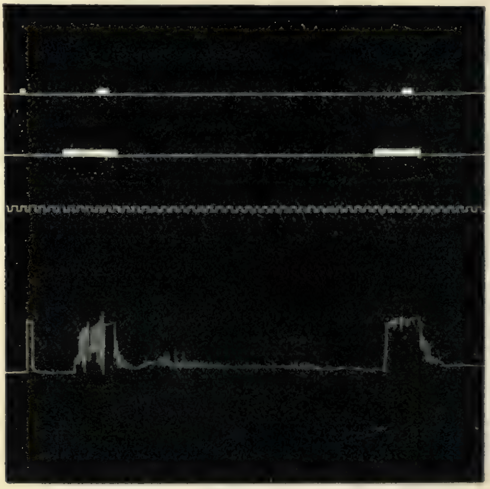

FIG. 8.

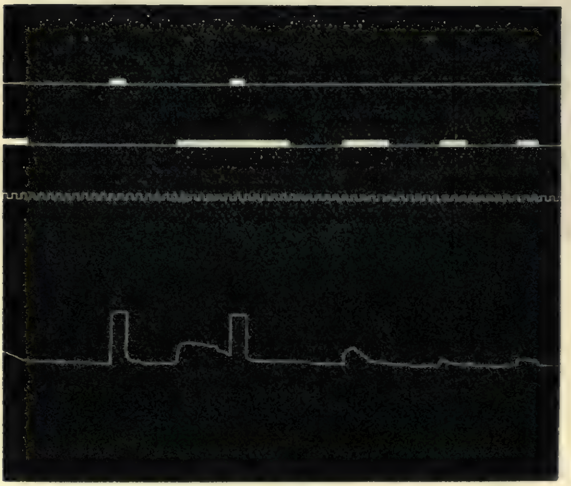

FIG. 9.

Figs. 7, 8 and 9.—These figures show the varying degrees of "lift' obtained from the cortex cerebri and motor nerve respectively and in association.

Fig. 7.—In this and succeeding photographs of tracings (reduced), the uppermost line marks by an electric signal

the moment and duration of excitation of the sciatic nerve; the second tissue similarly marks the excitation of the cortex cerebri, and later the spinal cord and central end of one sciatic nerve; the third line marks seconds; and the bottom line is the record of the weight attached to the muscle.

In other words, the nerve centres do not cause a complete explosion of the potential contractility of the muscle. Further, the average "lift" derived from the cortex is about three-fifths of that obtained from excitation of the motor nerve.

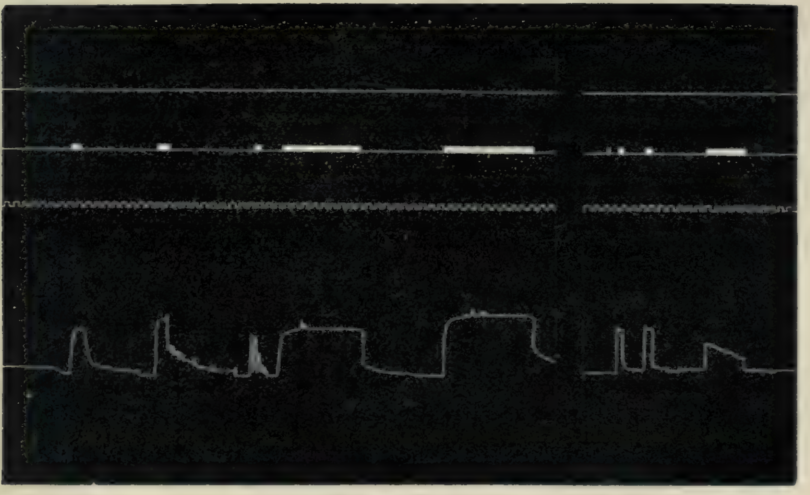

FIG. 10.

In an experiment conducted for the purposes of comparison such as I have described the cortex cerebri is at an advantage compared to the spinal centres, because clearly the order of events must be that to test the effect of the centres respectively on the same muscle the cortex phenomenon must be observed before the cord is cut for the purposes of obtaining in its pure form the discharge from the spinal centres. Thus the spinal centres in such an experiment are at great disadvantage, inasmuch for a time they are depressed by shock; but in this connection the hyper-excitability which soon supervenes and which was first minutely described by Franck,[1] counterbalances the error if the excitation results are taken at proper times after the section of the cord. To obviate this I have varied the experiment in different ways.

Allowing for errors in complications just referred to it would appear certain that the discharge from the cortical centre is on the average capable of evoking more work from the contracting muscle than a spinal centre, so that the centres should be arranged in that order cortex first and spinal cord second.

Thus I have performed some experiments in which the spinal cord was divided first, therefore no cortical excitation was undertaken, and only the spinal cord discharges examined with the result that a higher average "lift" was obtained.

Differentiation of the Effects.

(*a*) *Cortex or Cortical Effect.*—The character of the cortical discharge I have referred to, but I would like to point out that even at the present time the analysis of its components is very incomplete, and now propose to enter at once upon its analysis.

Firstly, the amount of energetic action by the centre, *i.e.*, demonstrated by the degree of the contraction of the muscle, varies inversely as Bubnoff and Heidenhain,[2] originally showed with the rapidity of the discharge of the centre, or, putting it in other words, with the shortness of the latent period. This fact is the experimental proof of Dr. Jackson's theoretical conclusions[3] on this subject.

In taking the whole curve of the muscle contraction from the cortical discharge we see at once that the greatest amount of work is performed as a rule shortly after the commencement of the excitation, that is, at the beginning of the fit; but this varies with the maximal or minimal character of the excitation. A minimal excitation produces a slow discharge of energy from the nerve centre (see fig. 11), and in accordance with the principles just stated resulted in a less

[1] *Loc. cit. supra.*

[2] *Archiv. f. d. Gesammte Physiologie.* B. xxvi., 1887.

[3] "Lumleian Lectures," *Brit. Med. Journal*, vol. i., 1890.

amount of work being executed early by the centre; whereas the maximal stimulus produces a high tetanic curve from almost the first moment, which is maintained at the same level for a short time, and then in respect of the maintenance of the application of the stimulus to be discussed presently gradually sinks towards the abscissa line. The moment the stimulus ceases, after a rapid diminution usually to or near the abscissa, there occurs a secondary gradual development of work done, and very rarely this after effect may amount to nearly the height obtained by the primary discharge from the cortex, *i.e.*, of the first stage. (See fig. 12.)

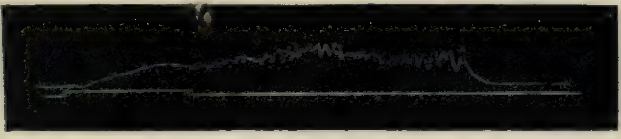

FIG. 11.

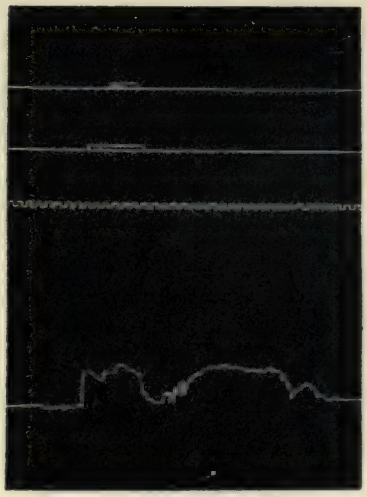

FIG. 12.

The clonic or secondary stage of the cortical effort usually attains to a certain height practically equal to that of an initial contraction, at least so far as the apices of individual clonic spasms, and this has been looked upon as a direct result of cortical activity. The character of its tetanic average is that of a gradual rise and an equally gradual fall.

The original experiment of Franck and Pitres, showing that when the cortex is cut off and the subjacent fibres of the corona radiata excited there is no clonic or secondary stage, has always been accepted as indicating that the clonic stage is entirely cortical, but actual proof was wanting until as stated above, the electrometer tracing showed that the clonic spasms were innervated by intermittent impulses of a similar rate and degree descending the spinal cord. It is now, therefore, clear that the clonic factor is of cortical origin, but the question remains, What is the source of the work done in the tetanic reinforcing curve of the clonic stage, is this also cortical or of spinal origin?

I have for some time been investigating this in relation to the whole subject of work done by nerve centres, and by varying the experimental procedure hitherto adopted have obtained some additional results.

It seemed to me that if there was this doubt, it could be to a certain degree cleared up if we could exaggerate the phenomenon itself, *e.g.*, by raising the excitability of the whole system, and especially the spinal cord. For this purpose in a certain series of experiments I stimulated, as shown in the tracing, the central end of the sciatic. Bubnoff and Heidenhain found that powerful excitation of the central end of a sensory nerve caused an increase in the amount of "nerve energy" put out by the cortex.

This we shall see is not always correct, and the exception reveals a very important condition of things.

In repeating these experiments in a slightly modified way I found that the tetanus of the *clonic* or secondary stage was increased when the ordinary tracing was taken and at last obtained in one striking instance the accompanying tracing (fig. 13), which shows what I wish to call a typical spinal cord effect occurring *after* the clonic stage, showing that at any rate in this instance if the spinal cord was in any degree responsible for the tetanus of the clonic stage it must be to a small amount. A similar tracing was obtained and figured by Franck,[1] but is not explained by him.

At this point I think it would be better to take up the question of the discharge from isolated spinal centres.

Discharge from Spinal Centres.—To compare the maximal discharge from the spinal centres with that obtained from the cortex or corona radiata it is of course necessary to employ an excitation evoking a tetanus; I have done this with the mechanical method, by taking a tracing of a simple reflex action and a crossed reflex.

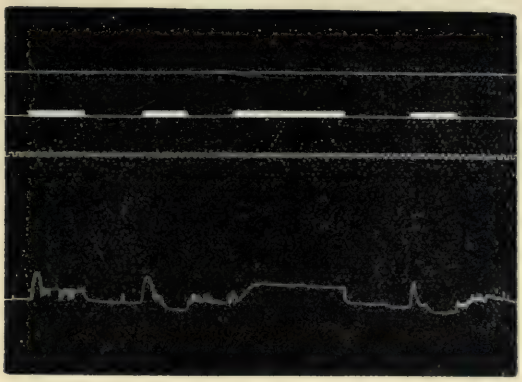

FIG. 13.

The typical "cord" effect is observed in the middle of the three curves as a prolonged tetanus after the clonic stage.

The simple reflex was obtained from the rectus femoris muscle through the right anterior crural nerve by excitation of the central end of the right sciatic, and the crossed reflex by the excitation of the central end of the left sciatic, the spinal cord being divided just above the middle of the dorsal region. In some cases a preliminary excitation of the cortex was first performed, but in others there was no interference with the parts above the section of the cord. The general results, *i.e.*, the work done by the muscle under these circumstances by simple and cross reflex action are as follows:—

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[1] *Loc. cit.*

We will first deal with the way in which the spinal cord responds to the simple reflex stimulus in order that we may define what is a characteristic spinal cord effect.

When the cord is excitable there is a very rapid response to the excitation, the weight is raised to the maximal height almost at once, and not infrequently there is an initial spike on the curve which may be on the ascending portion of the curve, or even when the curve has reached its maximum. This initial spike I propose to call the "initial effect" to distinguish it from a terminal spike to be described below (see fig. 14).

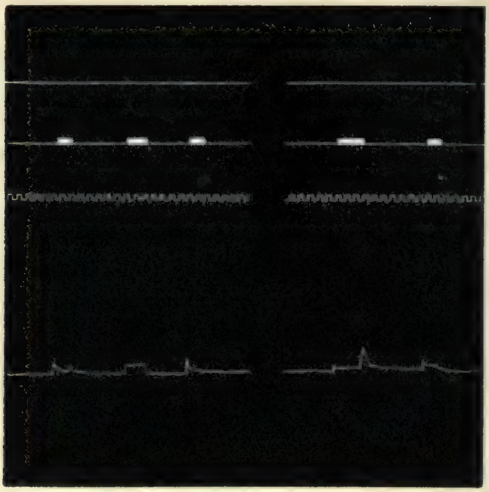

FIG. 14.

Simple reflex contractions showing double spike and large terminal spike.

It is not, however, necessarily of the same nature as the terminal spike, for apart from a genuine rapid contraction an initial spike may be drawn by the momentum of the apparatus. This, however, is easily distinguished from a real contraction rise. The line of the curve then runs horizontally forwards, more or less, and with indications of a rhythmical or clonic series of spasms (cf. Schafer & Horsley, *loc. cit.*), and with a more or less rapid descent, owing to "fatigue," in accordance with what has to be described directly, until we arrive at the end of the excitation.

In 1884 I had noticed frequently in the case of a spinal cord excitation, the occurrence of a spike at the end of the curve when the excitation was shut off, such spike resembling the large single clonic spasms often observed at the end of a cortical discharge. In 1881 Sanderson and Gotch noticed that when the spinal cord of a skate is caused to to discharge the electrical organ of that fish, it does so with an initial spike and a terminal spike. This form of discharge of the spinal centre must be, therefore, something of a very fundamental character if we find it ranging through the animal kingdom at such wide intervals as these; and here, again, it seemed to me that one might be able to bring the phenomenon into prominent relief by exaggerating the excitability of the cord and producing a premature dissolution of the same.

This phenomenon, which we may call the "end effect" of the spinal cord discharge, is closely associated with the question of the existence of a true spinal "after effect," comparable to the clonic stage or after effect derived from the cortex when the stimulus applied to the latter ceases. Without trenching on what has been said, I have now to point out that although in a more normal condition of the spinal cord, that is to say, during the early stages of an experiment, there is often no after effect to be obtained from the spinal cord resembling the clonic stage of the cortical discharge; still, an after effect may be produced at any time.

The after effect of the spinal cord, however, is so entirely different to the clonic stage of the cortical discharge that it is quite unmistakable, and when it is associated with the ordinary tetanic first stage derivable from the spinal cord, it is then so sharply defined as to justify the expression I used above of "typical spinal cord effect."

The form of the after effect varies with the intensity of the stimulus, as shown in the accompanying tracings.

A weak stimulus of 500 from the Kronecker coil gives a spike-formed after effect, which in many cases can be found to be a delayed end effect. I have found every transition stage between these. The usual spiked end effect simply requires to be delayed one or two seconds to be then an after effect. This is the first form of spinal clonic stage or after effect, and may be compared with the end clonic spasm of cortical discharge.

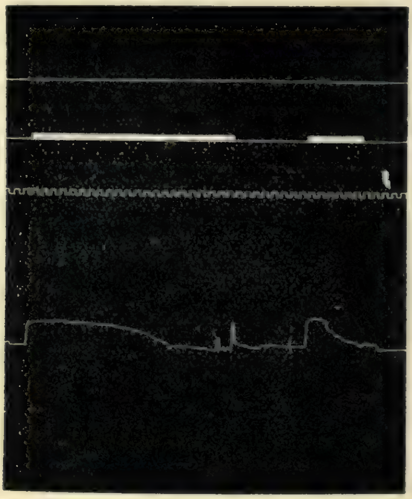

FIG. 15.

Excitation (reflex) of the spinal cord, showing difference of after effect, with (*a*) weak and (*b*) strong stimulus.

The second form is seen when a stronger stimulus (4,000 Kronecker coil) is used and when, as also will be seen directly, "fatigue" is showing itself in the nerve centres and assisting in increasing the delay, *i.e.*, intervals from two to six seconds. It, the after effect, begins slowly to reach a very moderate height and dies away slowly, sometimes exhibiting indications of a rhythm. (See fig. 15.) By taking a stimulus intermediate, say 1,000, one can obtain an after effect, the character of which is intermediate between the two just described. It is interesting to note that the difference in character of the after effects is not affected by exaggerating the excitability of the cord.

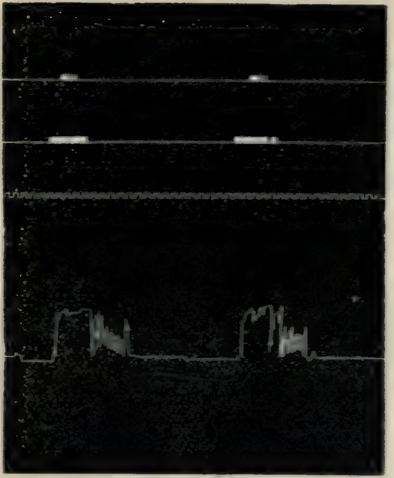

FIG. 16.

Cortex excitatory after effects to compare with the foregoing.

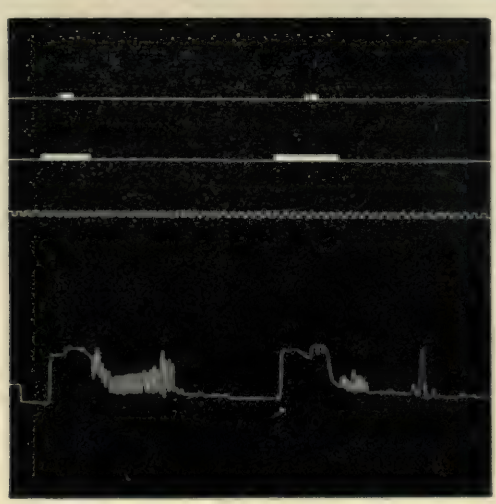

FIG. 17.

Cortex excitatory after effect; (*a*) common form; (*b*) delayed end effect.

No doubt with different conditions of dissolution of the cord, varieties more or less clonic in character could be obtained from the spinal cord if the exact moment was fortunately hit upon; a very exceptional case, for instance, was obtained by Schafer and myself in 1884 from the spinal cord of a rabbit. (*Vide Journal of Physiology.*)

We may therefore conclude that *any* nerve centres, whether in the cortex or spinal cord is capable of discharging (as would have been said some time ago "spontaneously") after it has been thrown into excitation by some definite stimulus, but that this after effect of the activity of the centre varies both in *character* and *force* according to the nerve centre producing it.

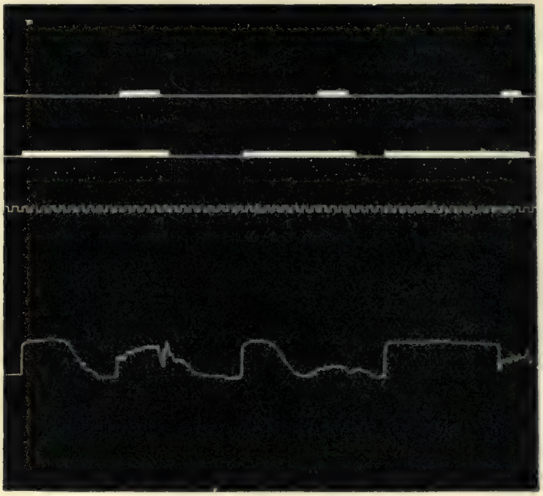

FIG. 18.

After effects varied by additional excitation of the spinal cord.

I now wish to show example in fig. 19 that a capacity for producing a second output of work as it were, can be demonstrated to occur during the discharge of the spinal nerve centre itself and is in effect one variety of Franck's *tetanos a renforcement*, as observed by that author to result from excitation of the cortex, this is very well exhibited in the dissolution of nerve centres by so-called fatigue and other depressing conditions, which subject I will now take up.

Effect of Disease State on the Output of a Nerve Centre.

Gotch and myself found[1] that the electrical effects obtained from nerve centres when the animal was suffering from some general pathological state, *e.g.*, from the severe disease known as distemper, were small. The same thing is true of course as regards the work done by the muscle

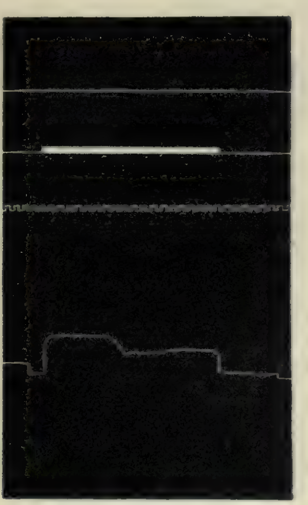

Fig. 19.

when the cortex is excited, and the character of the fatigue effect is that in accordance with the facts previously described the tetanus derivable from the cortex is very slowly developed soon fades down again to the abscissa line and that there is no tetanus in the clonic stage, the clonic spasms in fact simply start from the abscissa line and fall back thereto each time.

Another variety of the same thing in degree of discharge of the nerve centre under a hampering condition of this kind is that instead of a clonically marked tetanus the cortical discharge consists of a series of closely aggregated clonic spasms.

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[1] *Loc. cit. supra.*

As regards the spinal nerve centre discharge when affected by general depression effect, *e.g.*, disease, shock, etc., it is characterised by (1) great diminution of the work done; (2) slow development of the same and rapid failure.

Fatigue by Excitation and its Effect on a Nerve Centre.

Concerning the behaviour however of a normal nerve centre the most interesting phenomena are seen when it appears to be exhausted during the continual application of an electrical stimulus. It might be expected perhaps that by Pflüger's laws the increase of the excitatory stimulus would be followed by an increase in the work done by the nerve centre, but this is not the case, and the whole subject it will be found, is a complex and interesting one.

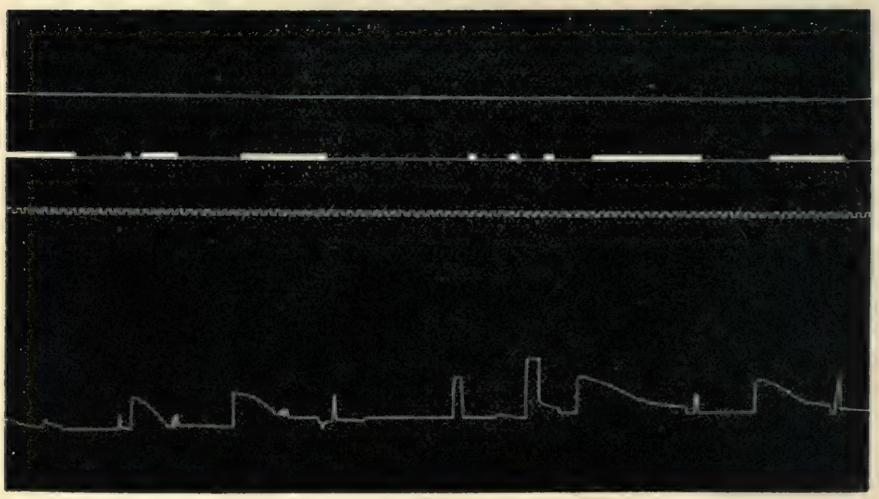

FIG. 20.

Fatigue effects on spinal nerve centres with strong stimuli. Note the spinal after effects.

Genuine Fatigue.

The easiest example is to take a reflex action. The following series of tracings show that with a stimulus of only 500 the degree of work done is very great. Prolonged excitation evoked continuous elevation of the weight, which is only very slowly and gradually lowered, whereas with an excitation of 4,000 the weight is maintained at its maximal height only for a short period and the descent to the abscissa line is very rapid.

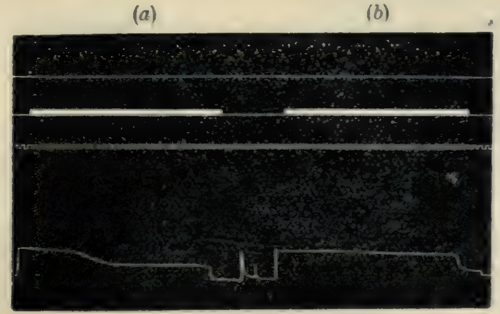

Fig. 21.

Comparison between fatigue of (*a*) reflex spinal centre; (*b*) fatigue of the muscle (on excitation of motor nerve).

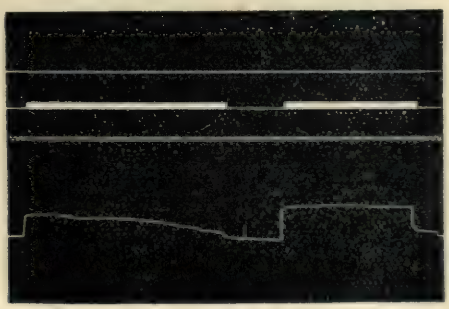

Fig. 22.

Fatigue of spinal centre compared with muscle; prolonged excitation.

I think it is questionable whether this is fatigue at all, or whether it is not rather an interference phenomenon, because if you throw into the cord an excitation of the afferent end of the opposite sciatic nerve during the fall of the weight towards the abscissa (the original excitation of the same sciatic nerve being continued all the while), it is found that there is a rapid and powerful raising of the weight and also a marked after effect of the kind characteristic of the spinal cord discharge when excited by a strong stimulus.

It cannot be said therefore that the spinal centre is exhausted in the ordinary sense of the word, but that the effect observed is more allied to the process of "inhibition."

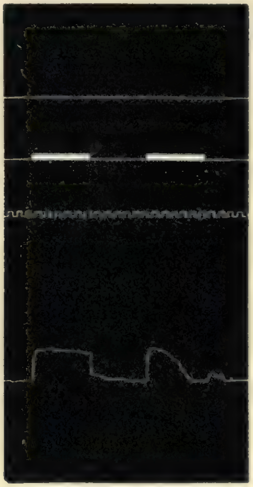

FIG. 23.

Effect of weak and strong excitation respectively on reflex centre.

To analyse this correctly, however, involves a review of our present knowledge of the arrangement of the antagonistic muscles in nerve centres. Attention has for some time been drawn by Sherrington, Beevor and others to the existence or non-existence of synergic action of antagonistic muscles, and has resulted in showing that when there is an active contraction of a flexor muscle, for example, that there is simultaneously an active relaxation of the extensor muscle.

This was first demonstrated by Sir Charles Bell and has subsequently been the subject of several papers. Several years ago Lowenthal discovered that excitation of the cerebellum evoked the same remarkable phenomenon, and the result of a long series of experiments by him and myself showed that this was due to the action of the cerebellum on the bulbospinal centres. In the same research we showed that the spinal centres themselves are arranged in an alternating fashion and that, for example, excitation of the central ends of similar nerves of the two limbs was followed by opposite motor effects, excitation of the central end of a nerve of the left side evoked a reflex contraction of a flexor, excitation of the same nerve on the opposite side produced a relaxation of that same flexor and contraction of the extensor.

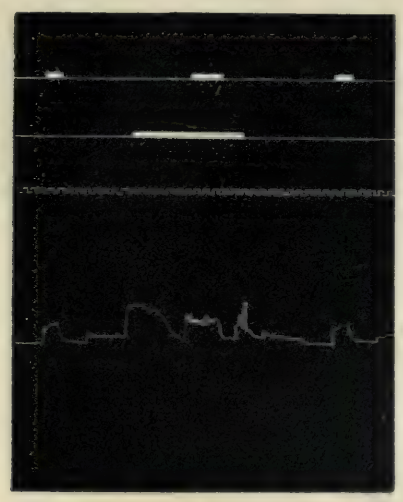

FIG. 24.

Combined excitation. The uppermost line indicates excitation of the central end of the opposite sciatic nerve. Powerful after effect.

Quite recently these observations have been amplified by Sherrington and Hering, and extended to an analysis of excitation of parts of the cortex cerebri. It is perfectly clear, therefore, that every kind of nerve centre at present capable of being isolated must be assumed to have the two opposite functions of antagonistic muscles represented in it, the further conclusion from which is that whereas one function may predominate over the other it is possible that an unequal excitation might produce a *confused* result, and might indeed go further, might convert a former contraction of the muscle into an active relaxation of the same. This latter would be called by some inhibition and by others might be translated as being due to exhaustion and premature fatigue of the muscle.

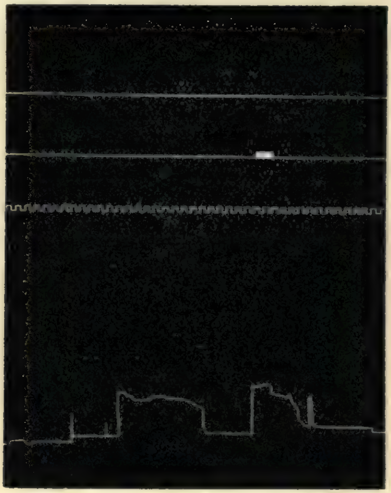

Fig. 25.

Comparison of after effect following strong excitation with absence of same after weak.

It was for the purpose of distinguishing between these two possible conditions that I have employed the combination of reflex excitation with the primary excitation, the results of which have certainly taken us far beyond what Pflüger's original laws would seem to indicate; and yet at the same time, as the following tracings show, there is, of course, much truth in their general idea that it is possible to raise the excitability of the spinal cord and get more work out of it. I can now show that the reciprocal innervation may be at the time being adverse, an example of this is shown in the case of a dog, where I found an unusual condition of things existing, namely, that excitation of the central end of the sciatic developed not contraction of the rectus femoris on the same side, but relaxation, just as though the hamstring muscle nerve had alone been stimulated (Sherrington), whereas excitation of the opposite sciatic nerve produced a small but well marked contraction of the muscle. This last effect, the crossed reflex, could be very notably increased by combining excitation of both nerves at once, showing that the general activity of the centre could be raised even under the adverse circumstances before referred to.

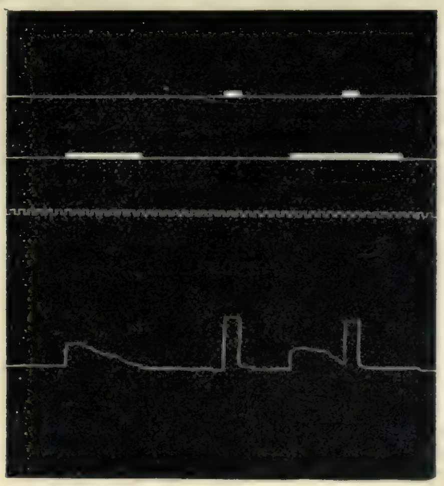

FIG. 26.

Excitation of motor nerve to muscle to show non-fatigue of muscle in contrast to the fatigue of the nerve centre.

Using the same considerations to analyse so-called failure of the cortex on excitation, the following tracings obtained from the cortex of a perfectly normal animal during a series of observations by Dr. Levy on the fatigue of the cortex cerebri, and for the loan of which I am indebted to him, show, I consider, the same state of affairs to be taking place, for the reason that the after effects are large and followed by the after excitation of the spinal cord to which I have before referred.

Herewith I also wish to record the occurrence of a very interesting delay of the onset of the clonic or after stage following excitation of the cortex cerebri, and which I have noted in cases where the nerve centres have been thrown into an abnormal condition by excitation of the central end of the sciatic nerve. See in fig. 28 the interval especially in the second example between the tonic and clonic stages. Such delay is explicable upon the ground that the opposite reciprocal innervation has been heightened by the abnormal stimulation of the centre.

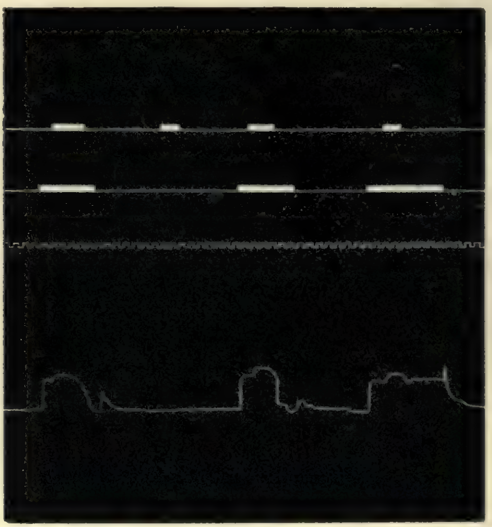

Fig. 27.

Excitation of both sciatic nerves (central end).

Correlative Phenomena Observed.

It will be remembered from what has been said above as regards the method of these experiments that the muscle employed has commonly been the rectus femoris, biceps and supinator longus, but most especially the first of these. During the progress of experiments I noted that the tail muscles exhibited in a very striking way exactly the same degree and kind of contraction as the rectus femoris muscle, but any tonic, *i.e.*, tetanic effect, was more prolonged in the tail muscles than in the rectus. So, too, occasionally, the afferent excitation of the sciatic caused rhythmical contraction of the rectum. Schiff long ago drew attention to the excitability of the centre in the spinal cord for the representation of tail movements.

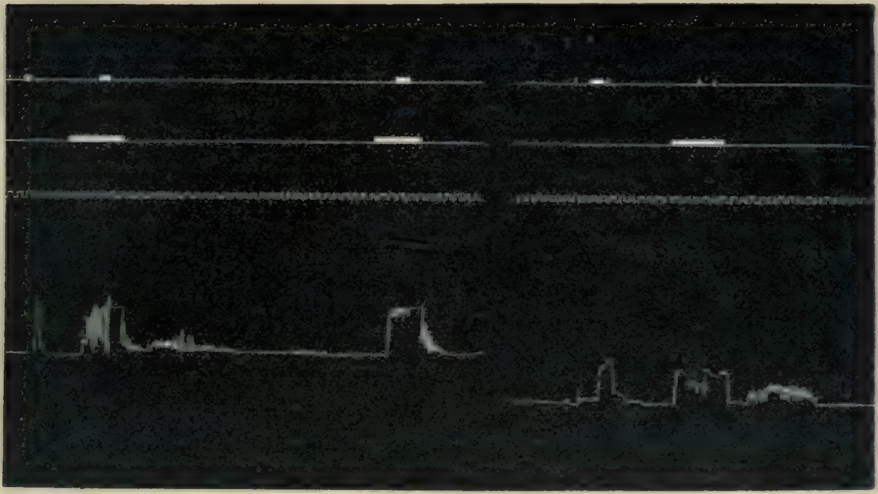

FIG. 28.

CASE OF EPILEPSY WITH TASTING MOVEMENTS AND "DREAMY STATE"—VERY SMALL PATCH OF SOFTENING IN THE LEFT UNCINATE GYRUS.

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P., LL.D., F.R.S.,
AND
WALTER S. COLMAN, M.D., F.R.C.P.

PART I.[1]

IN this article are given further particulars of a case of epilepsy which I published[2] about ten years ago. I will call the patient Z. There was in this patient's slight attacks the "dreamy state" (commonly called "intellectual aura"). With at least some of his seizures there were certain movements of the mouth and tongue, tasting movements; these were, I suppose, the indirect "reflex" results of an epileptic discharge beginning in gustatory elements of a certain region of the cerebral cortex—(Taste region of Ferrier.) After Z's slight attacks, or after some of them, there were very elaborate actions during "unconsciousness." The patient died, January, 1894, from an overdose of chloral. Dr. Walter Colman found at the necropsy a very small lesion of the left uncinate gyrus. A report of Dr. Colman's examination of the brain will be found in the second part of this article.

History of the Case of Z.

I first saw the patient, a medical man, whom I call Z, in December, 1877. He was then 26 years of age. He began to have slight epileptic attacks in 1871; they were so slight at first that he, to use his own words, "regarded the matter playfully and as of no practical importance." In 1874 he had a *haut mal*, and then for the first time knew the evil meaning of the slight seizures he had disregarded. He afterwards wrote out for me an account of his case, which, with his consent, I published (Brain, Part XLII., July, 1888, p. 201 *et seq.*). I may remark at once that there were no such signs of local intra-cranial gross organic disease (such disease as tumour, I mean) as optic neuritis and severe headache in any part of the patient's illness. I now give some details I obtained from Z in 1877.

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[1] This part is written by J. H. J.

[2] BRAIN, July, 1888. "On a Particular Variety of Epilepsy (Intellectual Aura); one Case with Symptoms of Organic Brain Disease." Case V. of that paper, p. 200, *et seq.* is the case of Z.

He had that variety of the dreamy state I call "reminiscence"; he called it "recollection." In his slight attacks there was, he told me, a sentence in his mind which was as if well remembered. For example, if anyone was at the time speaking to him it would be as if he (Z) were trying to remember it, as if it were familiar, but yet he could not remember it. Again, he said—I give the words I hastily wrote in my case book, here intercalating other words in square brackets—"Attending to what was going on in [my] mind because [it was] interesting, and dim to what [was] going on outside." He could not, on recovery, remember what the "interesting matter" was. (*See* Brain, July, 1888, pp. 202-3, for further particulars of his dreamy state.) According to a report Z gave me in 1890, the reminiscence had disappeared, and from this report I gathered that he had had another kind of dreamy state with the reminiscence, and that this other kind occurred in his later fits. After the severe fit in 1874, in which he bit his tongue, he was subject both to slight and to severe attacks.

There was no evidence of any crude sensation warning of smell, taste, hearing or sight, nor did I ever afterwards hear of any; there was no epigastric sensation. There was no vertigo. Although there was no crude sensation warning of taste at any time, there were certain movements which, I think, imply an epileptic discharge of gustatory elements of the cortex cerebri. I did not, when I first saw the patient, obtain any information as to such movements.

He wrote of them (BRAIN, *op. cit.*, p. 204) as being, he had been told, "*somewhat like a modified and indistinct smacking of the tongue like a tasting movement, and is generally accompanied by a motion of the lower jaw* (italics in original), and sometimes by some twitching of the muscles round one or both corners of the mouth or of the cheeks, *but by no sense of taste in my recollection*." (My italics.)

I ought here to say that several observers, one of them a highly accomplished medical man, who saw many of Z's attacks, never noticed any such movements as I have mentioned. They were very slight; in an attack (*vide infra*) which I witnessed, the noise made was only just audible. That they occurred in at least some of Z's slight attacks I do not doubt, because of the observation I made, and because he reported (BRAIN, *loc. cit.*, p. 204) that some others had noticed them. It may be that the patient had, during the period this medical man knew him, ceased to have such movements. Z wrote to me, May, 1890, a report in which, among other things, he says, comparing with previous years: "Although I have often asked observers to describe such a sound as I have made, and which in former notes [those published, BRAIN, July, 1888, p. 204] I described as a smacking of the tongue like a tasting movement, I have not found it so described, but rather as some indistinct attempt to speak."

He had a slight feeling of dread. It was not a fear of the fit; the dread came first and then the fit, or rather the rest of the fit.

He said the fit seemed somewhat within his control; he made a rule when it set in to try to do something definite, for instance, to speak. He gave, as another example, that he once made an excuse to pick up a ball. In August, 1880, he was still subject to slight fits. When a clinical clerk, he would have them in the wards of the hospital, but they were so slight that no one noticed anything particularly wrong. One medical man observed something peculiar, and so did several friends. Z was told that he appeared anxious to answer, but seemed to have lost the drift of the conversation—used in the connection some wrong word, and yet appeared to search about for another word. This is not very definite, but serves to show how slight the slight attacks were at that time.

In the remainder of this article, I shall speak only of Z's slight seizures. I witnessed but two of them, and these I saw years after his first visit to me. In one he stopped talking to me, remained standing, and made slight, very slight, just audible (*vide supra*) smacking movements of his lips. I have no further details of this seizure; it occurred at the meeting of a Medical Society, and I had no opportunity of watching him throughout the attack. I knew that he was quite safe; no one but myself noticed anything amiss with him. On another occasion he was sitting in a room consulting me; he stopped talking—I have no remembrance of any smacking movements of his mouth on this occasion—his head was bent forward, but in a second or two, the paroxysm being then, I suppose, over, he looked up, and next (post-paroxysmal stage of actions) he leaned over one arm of his chair and felt about on the floor as if searching for something; next he did the like on the other side. Shortly, having a pin in his hand (how he got it I know not, perhaps from the floor) he made a feint of pricking my hand; the action was as if in fun, for he stopped well short of my hand and was smiling. This little affair was exactly after the manner of joking with a child, as if one said playfully to a child, "Now I'm going to prick you," and smilingly pretended an attempt to do so. Z, however, did not accompany his playful-seeming feint by words. Soon, perhaps a minute, afterwards, his actions, or I should say the irrelevant-seeming actions, ceased; he replied correctly to simple questions, and told me that it was not necessary for me to go home with him. He, however, looked confused and seemed strange. When we got to his house a few yards away, I thought he was fully recovered, and, as I was thinking of making another room on the ground floor of my house, I took the opportunity of speaking to him about a third room there was on the ground floor of his house. Among other things he said he used to breakfast there. I was surprised when he afterwards, next day, told me that he remembered nothing from the time of being in my room consulting me (before the fit) to a little time after I left him at his own house.

These post-paroxysmal actions during what we clinically call unconsciousness were as elaborate and purposive-seeming as any of those of his normal self; such post-paroxysmal actions are very important in a medico-legal point of view. On another occasion there were post-epileptic actions by Z during "unconsciousness," of a kind which in a man fully himself would be criminal, and must have led to very serious consequences had not, fortunately, his condition been known. What he did was overlooked by those concerned.

In Z's report of his case (BRAIN, July, 1888, p. 206, paragraph 4), he speaks of examining and prescribing, when temporarily unconscious after an attack, for a patient. I now give a specimen of what he wrote—notes of a patient's case—in such circumstances. The words I have put in curved brackets were crossed out by Z; those in square brackets are my words. "For six or seven (years) weeks he has felt very discomfort of the throat and also for the swelling of the throat and (legs) arms and ——" [The word for which I have put a mark signifying a blank I cannot decipher]. Here is another specimen: "For the last few days his beginning (starting to walk ?) is more difficult for his tenderness of speechlessness and quick power of talk light swolleness of feet last three days." The words "starting to walk" in the square brackets were inserted later. Here is another specimen with Z's remarks on it: "I had written the above [*i.e.*, he had filled up the form for name, age, &c., of the patient] correctly and ascertained that the man had had gradually increasing œdema of the legs lately, when a *petit mal* came on with no overpowering impression, or indeed any distinct impression that I can recollect. I wrote the words below [below the heading] slowly and with difficulty, wishing to be correct; on reading them over they seemed to me correct; but I could not go on; I turned over the page and tried to describe the man, stared at him and felt too undecided to write more than 'A rather';—could easily copy what I saw before me written down over the man's bed already as to the urine. Altogether it took two or three minutes. I remained undecided and disinclined to write, but able to talk for five minutes more." This is what Z wrote on this occasion: "For about the last fortnight about the legs are about the gradual for several debts of the" (Z adds, "no connection in thought traceable for the word 'debts'").

Still one more specimen of Z's writing. He wrote the following in a state, which at the time, he thought was full consciousness, but afterwards knew to have been defective consciousness: "There [then a word obliterated] was constant repetition of sickness for the last twenty-four hours. Abdomen [this word crossed out]. The sick [a word like 'neck,' joined to sick, crossed out] on the grateful rightnessness has felt a large knowfulness." Z wrote down when well what he thought this should have been: "The sickness has left a painful feeling on the right side; she has felt much fulness there."

The following specimen of Z's writing (it was written as notes of a case) was not corrected: "For the last 18 mos [mos, no doubt, is for months] years there has been some decided indefinite on R. side in dress circle." Moreover in the heading of the case in the part for occupation he made a mistake—"Hairdresship" for, I presume, "Hairdresser." The patient was a woman, but this may have been for her husband's occupation.

For localisation of the lesion I relied entirely on Ferrier's researches. I say this not only because credit should be given to the right man, but also because, having said what I have said, I can without immodesty put some facts more strongly than I otherwise should like to do. Let the following four statements be considered.

(1) Ferrier found that certain movements of the lips, tongue and cheek-pouches follow on artificial excitation of a certain region of a monkey's cortex. From the nature of these movements he inferred that that region contains gustatory nervous elements—that the movements were "reflex" consequences of the artificial excitation of those

elements. (2) I suppose that similar, or analogous, movements in human beings subject to certain epileptic fits, as in the case of Z, at the onset of their seizure, are "reflex" effects of epileptic discharges beginning in some gustatory elements of that cortical region which, according to Ferrier, is for taste. (3) I begged Dr. Colman to call on me before he went to make the necropsy on Z, in order to ask him to search the taste region of Ferrier on each half of the brain very carefully. (4) Dr. Colman (see Part II.) found a very small focus of softening in that region (in the uncinate gyrus) of the left half of the brain.

There was no trustworthy evidence to point to the half of the brain affected. Z wrote (Brain, July, 1888, p. 204): "I have no clear evidence that one side of the face is affected more than the other, and no clear evidence against it; from what little I can learn, if it is at all unilateral it is more on the right side than on the left; but the evidence is very scanty. I never notice it myself. I also never notice myself, but learn from others, that sometimes, especially if sitting, I give one or two light stamps on the floor with one foot, and in the only cases where this has been accurately observed it has been with the right foot."

There are many points for comment in Z's case. I refer to "Lectures on Epilepsy," *Medical Times and Gazette*, vol. i., 1879; Brain, July, 1880; Brain, July, 1888, for remarks on the group of cases to which this case belongs. In those papers I consider the Dreamy State in its association with crude sensations of smell and taste, and with the epigastric sensation and with chewing and tasting movements. In Brain, October, 1889, Dr. Beevor and I record a case of epileptic attacks with a crude sensation of smell and a dreamy state; there was tumour of the right temporo-sphenoidal lobe. In that paper Dr. Beevor refers to cases previously recorded of epileptic attacks with warning of smell. In the *Lancet*, January, 14, 1899, I published a note on what I call the Uncinate Group of epileptic fits. I there consider the asphyxia in slight fits of some cases of this group, and suggest, having regard to some researches of Mr. W. G. Spencer (*Transactions of Royal Society*, vol. clxxxv., 1894, B, pp. 609 to 657), that the asphyxia may be owing to inhibition of the respiratory (medulla) centre by discharge spreading from a discharge lesion of the uncinate gyrus to the cortical centre for arrest of respiration; this arrest centre is close in front of the uncinate gyrus.

PART II.[1]

Pathological Report.

The autopsy was made thirty hours after death. The weather was very warm and damp. *Post-mortem* lividity was extreme, and decomposition was rather advanced.

Examination of Head.—The scalp was unusually thick, and firmly bound down to the periosteum. The *skull cap* was natural except for two symmetrical, flat, rounded elevations about half-an inch in diameter situated on the outer table, on each side of the bregma. The *dura mater* was unusually adherent to both parietal bones, but not elsewhere. The surface of the pia-arachnoid was normal, except beneath the bony prominences at the bregma, where there was some adhesion. Pacchionian bodies were large and numerous in this region, but inconspicuous elsewhere. There was no adhesion between the pia mater and the surface of the convolutions at any part.

Both hemispheres were fully convoluted, especially in the frontal and occipital regions, the convolutions following the usual arrangement. Their surface was perfectly normal in appearance, and the consistence of the brain was natural except in the left uncinate gyrus, where it could be felt that there was a patch of softening beneath the surface. The position of the softened patch is shown in fig. 1, which is drawn from a tracing of the brain after hardening in spirit. On a section being made through the patch it was found to be a small cavity, collapsed and almost empty, with indefinite walls, situated in the uncinate gyrus, $\frac{3}{8}$ inch below the surface just in front of the recurved tip of the uncus (*see* figs. 1 and 2). The existence of the patch

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[1] This part is written by W. S. C.

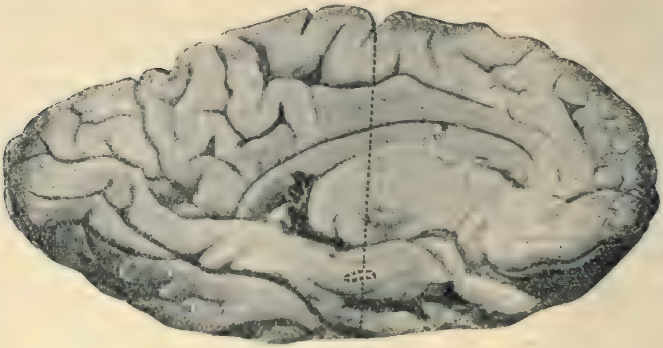

Fig. 1.

Drawing of internal surface of left hemisphere (case of Z). The black ring corresponds with the position of the cavity found, which is shown in section in Fig. 2.

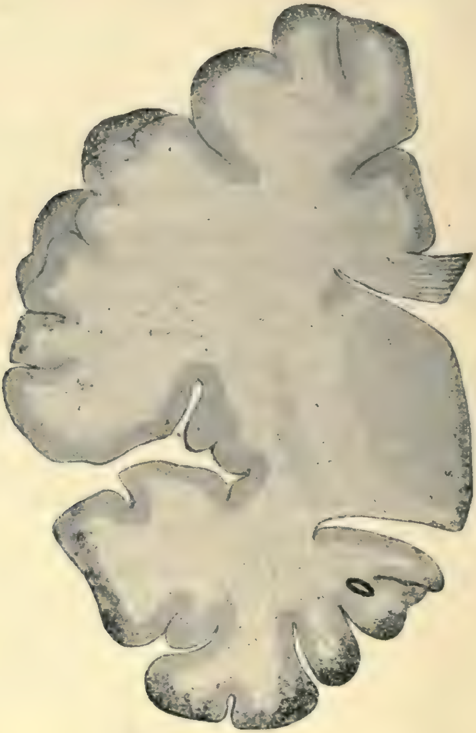

Fig. 2.

Vertical section of left hemisphere (case of Z, from a tracing). Site of small cavity apparently due to softening, shown by black ring.

and its position were verified in the fresh specimen by Dr. Hughlings Jackson, Dr. Dawtrey Drewitt, Dr. James Taylor, and Dr. Guy Wood. The cavity was like those seen long after softening from thrombosis or embolism; there was no surrounding inflammation nor any indication that it was of recent origin. Search was made for an occluded artery in the neighbourhood, but without success. The other vessels of the brain did not show any signs of atheroma to the naked eye.

Unfortunately the uncinate region became so soft and friable during the hardening process that it was impossible to make satisfactory sections, and the microscope did not throw any further light on the cause of the lesion.

The urine contained a small amount of albumen, and there were early interstitial changes in the cortex of the kidneys.

There were many pulmonary infarcts apparently produced during the prolonged coma which was present for twenty-four hours before death, and the lungs themselves were extremely œdematous.

With these exceptions the organs were healthy.

A localisation of the lesion in this situation had been suggested by the presence of the "dreamy state" or "intellectual aura." In a paper read before the Medical Society of London in 1889 and afterwards published in BRAIN, Drs. Hughlings Jackson and Beevor published the case of a woman, 53 years of age, who had a tumour, the size of an orange, at the anterior extremity of the temporal lobe. During life she had numerous fits, with an intellectual aura and with "a horrid smell." A very instructive case recorded by Sander[1] is quoted in the paper by these authors. The patient had fits which were preceded by the warning of a "dreadful disagreeable smell," and he then had "*chewing movements of the jaws, and spitting of saliva*." Later on he had convulsions about the face, but not in the limbs. He became very dull mentally, so that few subsequent observations on his sense of smell or of taste could be made. On *post-mortem* examination a gliomatous tumour the size of half a large apple was found on the under surface of the brain, at the junction of the left frontal and temporo-sphenoidal lobes. Half the growth involved the anterior part of the temporo-sphenoidal lobe, the other part reached across the fissure of Sylvius involving the lower convolutions of the frontal lobe. Inwards the growth reached the middle line.

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[1] Sander, "Epileptische Anfälle mit subjectiven Geruchs Empfindungen bei Zerstörung des linken Tractus olfactorius durch einen Tumor." *Archiv f. Psych.*, vol. iv., p. 234.

Reviews.

Die Bedeutung der Augenstörungen fur die Diagnose der Hirn- und Ruckenmarks-Krankheiten. Von Dr. OTTO SCHWARZ, Privatdocent an der Universität, Leipzig. ("The meaning of disturbances of the eyes in the diagnosis of disease of the brain and spinal cord.") 1 vol., 8vo, pp. 100. Karger, Berlin. 1898.

FOR the past few years the interest in the connection of ophthalmology and general medicine has been steadily increasing, and has found its expression in the appearance of several works. Amongst others, the valuable book of Berger ("Les maladies des yeux dans leurs Rapports avec la Pathologie Générale,") and that of Knies ("Die Beziehungen des Sehorgans und seiner Erkrankungen des Körpers und seiner Organe"), treat of the relationship between eye disturbances and diseases of the nervous system. In these, however, the eye conditions are not viewed from the standpoint of neurological diagnosis, and in many handbooks on neurology and general medicine, they do not hold the important position they deserve. Dr. Otto Schwarz, therefore, feels justified in presenting this work, dealing more exactly with the meaning of eye trouble in the diagnosis of disease of the brain and spinal cord. He claims a certain authority in so doing, having had peculiar advantages for systematic work in this direction for many years. His endeavour has been to give to the neurologist the clearest possible picture of the signification of eye disturbance, and to assist the ophthalmologist in forming a comprehensive opinion, so that he may be of assistance to his medical brethren in the diagnosis of nervous diseases.

This work is a valuable one, in that it is an honest, capable effort to bring into concise and simple order the items of a very difficult and, it must be confessed, little known subject. Years of careful and progressive observation must pass before much certainty in this remarkably abstruse and peculiarly elusive field can be obtained.

There is no doubt that the author has built up his book as thoroughly as is possible, and has taken his facts from the most reliable sources. It is in some places laboured, not easy of expression, and as disconnected as a dictionary. Possibly this is not to be avoided to a certain extent, owing to the great difficulty of obtaining sound and sufficient information in some of the more or less rare diseases of the nervous system.

About twenty-eight pages out of the hundred which make up the book are given to Hysteria, and are remarkably good and instructive. This undue proportion is explained by the author as being due to three reasons: (1) the great variety of the symptoms; (2) that no systematic work, from the diagnostic point of view on this disease, exists in the German language; and (3) that the writer has had long and rich experience of the malady. The author has not been able to resist the temptation of giving us a new definition of Hysteria, which may or may not add to our power and facility of appreciating this abnormality.

As a work of reference this book will be very valuable, and should be in the possession of every neurologist.

H. WORK DODD, F.R.C.S., &c.

The Nervous System and its Diseases. By CHARLES K. MILLS. 1 vol., large 8vo, pp. 1056. Pentland. 1898.

THIS is the latest of the large text-books on neurology which have been given us from the American continent, and it is, without doubt, the most extensive which has appeared in the English language since the monumental work of Sir William Gowers.

The volume is complete in itself, in so far as completeness is possible in a rapidly progressive subject; a copious bibliography and general index putting the reader in possession of the latest publications bearing upon the anatomical, physiological and clinical relations of neurological work.

The first portion of the book treats of the anatomy, physiology and chemistry of the nervous system. Into this part has been incorporated the terminology and nomenclature introduced by Prof. Wilder, of Cornell. Whether this terminology is likely to receive that general recognition and adoption which the author hopes, is a point on which some doubt may be thrown. However desirable general conformity in nomenclature may be, it will be long before such classical terms as "foramen of Monro" and "lateral ventricle" are known as "porta" and "paracoelia" respectively.

The second portion of the work is devoted to various general considerations, such as etiology, methods of investigating cases, the practical applications of electricity, and the general therapeutics of nervous disease; which chapter is from the practitioner's point of view one of the best in the volume. Here we have detailed the important subjects of balneology, massage, systematised movements, and the "rest cure." Several pages also are given over to the uses and applications of drugs in the treatment of nervous disease.

Four hundred pages are given to the consideration of the organic diseases of the brain and its membranes. It is impossible, in this short review, to follow the author through the mass of valuable material which he has brought together to throw light on this subject. Suffice it to say that there is no known condition which has not received ample consideration. More especially would we refer to cerebral localisation in all its bearings, the chapters on which appear to be the most complete with which we have met.

In the remainder of the volume the affections of the special sensory, and of the motor cranial, nerves are considered. The book would occupy in neurological literature a position of preeminence were this the only subject of study. They are the most detailed and comprehensive which we have come across in our reading. Were it not invidious to draw comparisons between chapters, all of which are valuable and pregnant with facts of interest, we would particularly refer to those upon the special senses as being worthy of the closest study.

The reading of this volume encourages the hope that the author will give us the other, which shall include the remaining diseases of the nervous system, insanity, and the medical jurisprudence of nervous and mental disease, which he has conditionally promised in his preface.

W. ALDREN TURNER.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

June 30.—Dr. C. E. **Beevor** opened a discussion on "The Accurate Localisation of Intra-cranial Tumours," and Drs. **Byrom Bramwell**, **Mitchell Clarke**, and **Sharkey** took part in the discussion.

At the Clinical Meeting of the Society, held at 11, Chandos Street, W., on Thursday, October 27, 1898, at 8.30 p.m., the following cases were shown :—

Dr. **Ormerod** and Dr. **Batten**.—Idiopathic muscular atrophy (Landouzy-Dejerine type) in mother and son.

Case I.—M. S., 39, ten years ago first noticed weakness of left arm ; both arms became gradually affected ; five years ago began to experience difficulty in going up-stairs. No alteration has been noticed in the face, but the patient states that when a child, her mother did not like to go into the room in which she slept as her eyes were not closed during sleep, and seemed always to be looking at her.

Family History.—Married ; three boys ; no miscarriage. First child described below. Second and third boys said to be healthy. No history of a similar affection can be obtained in her family or their children. She has five sisters and two brothers.

Present Condition.—Facial atrophy ; weakness of orbicularis oris and palpebrarum ; atrophy of trapezius and rhomboids ; "winging" of scapulæ ; weakness of anterior tibials and peronei of both legs ; pes cavus. K. Js. active ; no A.C. ; plantar slight.

Case II.—E. S., 5 years ; never able to close the eyes well and sleeps with them open ; two years ago had his canaliculi slit at Moorfields in consequence of tears falling over his face, and on one Christmas previous to this he was presented with a trumpet which he was unable to blow until his mother held the corners of his mouth.

Present Condition.—Very marked weakness of frontalis and orbicularis palpebrarum and oris; weak zygomatici; body thin; all movements capable of being performed, but some weakness of anterior tibials and peroneal muscles. K. Js. present, equal.

Dr. BEEVOR.—Unilateral wasting and paralysis of lower half of trapezius.

Patient is a girl, aged 20, who had typhoid fever and "brain fever" at 12 years, and nine months after her scapula was noticed to protrude. At present there are no fibres of the trapezius remaining below those which pass from the second dorsal spine to the inner end of scapular spine, and the rhomboid is visible. No other muscles affected.

The want of action of the muscle is seen in advancing or abducting the arm to the horizontal line, but beyond that point the serratus magnus acts.

The condition is probably a neuritis or a poliomyelitis.

Dr. H. SINEGAR.—Case of idiopathic muscular atrophy.

A female, aged 26, noticed weakness in her right arm eleven years ago, her attention being first attracted to it by difficulty in doing her hair. Wasting and weakness soon involved the left arm and steadily increased on both sides. She has been unable to put her hands on her head for five years.

Family History.—Negative.

Present Condition.—Weakness of orbicularis palpebrarum; winging of scapulæ; wasting of shoulder and arm muscles. Muscles affected; on right side, supinator longus, pectoralis major, serratus magnus and rhomboids practically absent, biceps and trapezius markedly wasted; on left side biceps, supinator longus, serratus magnus and trapezius practically absent, triceps and rhomboids greatly wasted. The deltoids and supra-spinati are distinctly hypertrophied on both sides. K. Js. active; no ankle clonus.

Dr. F. W. MOTT.—Arsenical neuritis from local action of the poison followed by general degenerative effects upon the nervous system.

A temperate man, aged 56, who had never had any illness before, was employed at chemical works to scrub the sheep-dipping compound from the indiarubber clothes of the men employed in its manufacture. This compound contains a large quantity of arsenic and alkali. About six weeks after he had been at the work he noticed a numbness and tingling in the fingers of the right hand, with gradual and progressive loss of power, so that he was obliged later on to give up the work because he could not hold the scrubbing-brush. He had considerable loss of sensation to pressure, heat and cold over the right forearm and hand, and he was unable to use his hand. The reaction of the muscles to the faradic current was diminished. He had had no general symptoms of arsenical poisoning.

Six months later he came complaining of trouble in passing water, and although sensation in the arm and hand had improved, there was considerable loss of power, and the skin of the hand was smooth and glossy. To the local condition, however, there were added new symptoms, viz., paresis in the lower limbs with spastic gait and very exaggerated knee-jerks.

While the hand has somewhat improved so that he can feel a light touch, the paresis and spastic gait has increased, and he has, therefore, been admitted to Charing Cross Hospital.

Dr. Ormerod.—Hereditary Chorea. A woman aged 50. Subject to constant irregular movements of the face, trunk, and limbs, which in character recall those of a mild chorea. No other physical abnormality. No marked defect of intelligence, but she appears to be rather forgetful and garrulous.

The movements began two and a half years ago, after a shock; she had been dismissed and forcibly ejected by her employer.

No previous chorea. No rheumatism.

Mother died of a similar complaint, aged 57, after fifteen years' illness. *Mother's mother said to have had the same* disease. No insanity known in her family.

Of eight brothers and sisters only one survives (a sister aged 52, healthy). Of the rest, two brothers died in early manhood, and the rest quite young. None had this complaint. Of six children, three died in infancy, *one aged* 20, *of rheumatic fever*, two survive and are in good health.

Mr. Lake and Mr. Ballance.—Case of recovery from temporosphenoidal abscess.

Mr. D., aged 33. Long standing left otorrhœa with sudden onset of intra-cranial symptoms; namely, headache, vomiting, slight right hemiplegia, pupils dilated, sluggish, left optic neuritis, no hemianopia, partial aphasia, alexia, and agraphia, complete left facial palsy, copious purulent flow from left ear, pulse 44, temperature 98° F.

January 26, 1898. *Operation.*—Pus seen pouring through attic. ℥ii. pus evacuated from temporal lobe. Drainage of abscess three weeks. Complete recovery.

Dr. Beevor and Mr. Ballance.—Case of removal of subcortical cerebral tumour.

Mrs. S., aged 39, admitted into National Hospital, May 21, 1894.

Symptoms.—Headache, vomiting, optic neuritis, aphasia, partial right hemiplegia, right hemi-anæsthesia of cortical type.

First operation, July, 1894. Large infiltrating mixed-celled sarcoma, removed from upper part of left parietal region.

Re-admitted February 22, 1897. Almost complete aphasia—more paralysis, headache, &c.

Second operation, March, 1897. 5⅓ ozs. of tumour removed.

July, 1898. Left hospital able to walk alone; no aphasia, &c.

Dr. TAYLOR and Mr. BALLANCE.—Case of Trigeminal Neuralgia.

Mrs. N., aged 66, ten years ago commenced to have attacks of pain in right side of face. Meckel's ganglion removed five years ago. Pain absent fourteen months; then suddenly recurred. Last three months violent exacerbations affecting all three divisions of right fifth. Admitted National Hospital, August 10, 1898. Urine contained trace of albumin. Operation (Krause's) in three stages, August 18, 22, and 26. Result: complete relief from pain; taste sensation on posterior part of right side of tongue abolished.

Dr. LUNN and Mr. BALLANCE.—Case of removal of cerebellar tumour, three years and eleven months after the operation. The case was shown before at a meeting of the Society eighteen months ago.

At the Pathological Meeting of the Society, held at 11, Chandos Street, Cavendish Square, W., on Thursday, December 15, 1898, at 8.30 p.m., the following communications were made:—

Dr. MOTT.—*Changes in the brain, muscles, and other organs, found in persons having died with prolonged epileptiform convulsions.* The brain and spinal cord were examined by the Nissl and the Weigert iron methods in the following cases:—

(1) Boy, who died probably from sunstroke; death preceded by 22 fits and a temperature of 108° F. (2) Thrombosis of the great anastomatic veins of Trollard, occurring first in the left hemisphere, then in the right. The patient had a succession of right-sided fits, then a succession of left-sided fits; he died comatose with a temperature of 107·5° F. (3) Three cases of *status epilepticus* occurring in young and previously physically healthy individuals. Death ensued from asphyxia and heart failure, with enormous venous engorgement of the organs. (4) Case of rupture of middle meningeal artery on the right side followed by over

400 left-sided fits; the patient was trephined but died shortly after. (5) Case of a middle-aged man, said to be an epileptic; died after seven fits. *Post-mortem* discovered advanced chronic Bright's disease. ? Convulsions uræmic. (6) Spinal cord of a case of general paralysis; temperature 109° F., death. *Post-mortem:* hæmorrhage from spinal veins, causing hæmorrhagic pachy-meningitis spinalis universalis. (7) Case of septicæmia with prolonged pyrexia, terminating in hæmorrhages and a very severe anæmia, so that the blood showed an enormous number of nucleated corpuscles, and yet the cortical pyramidal cells appeared normal.

In the cases 1 to 3 inclusive, a very similar condition of things was found; namely, venous congestion, stasis, and minute hæmorrhages of the brain, and under the pia mater. Œdema with distension of the perivascular and perineuronal lymph spaces; arterio-capillary anæmia and marked changes in the chromatic substance of the cortical pyramidal cells.

In some instances, owing to the distension of the lymphatic spaces, a continuation can be made out between the lymph space around a capillary and the lymph space in which a cell lies. It would thus appear that there is a canalicular system of lymphatics in the brain.

Owing to the distension of the lymph space, it is impossible to say whether the cell is apparently or really shrunken. The nucleus is usually enlarged, and the whole cell stained a uniform colour; the normal Nissl bodies cannot be seen either on the protoplasmic processes or in the body of the cell. If the section be stained with polychrome blue or methyl blue and safranin, then examined with a high power, it will be observed that the body of the cell consists of a fine net-work stained pink. Along these delicate pink lines, extremely fine, blue particles can be seen encrusted—the remains of the chromatic substance at the nodal points of the trabeculæ look coarser, owing to aggregation. The normal spindle-shaped chromatic bodies on the protoplasmic processes have either altogether disappeared, or are extremely attenuated. The only part of the cell which retains its normal blue stain is the nucleolus. These cells therefore present a marked contrast to the cortical pyramidal cells of Case 7. Examination of the anterior horn cells of the spinal cord, and of their homologues in the medulla was not made in Case 1, but in all Cases under 2 and 3 comparatively slight changes only were made out. These results agree strikingly with observations which I made of the cortical pyramidal cells of monkeys, in which Dr. Leonard Hill had ligatured two carotids and one vertebral artery, and show that local rather than general conditions were the cause of the marked changes observed in the cortical pyramidal cells as compared with the spinal motor cells.

We will now consider the general causes which might occasion the changes. (1) Pyrexia: This might have been the cause in Case 1, because the cord was not examined; but in the other cases the marked changes described in the cortical pyramidal cells were not found in the spinal cells. The anterior horn cells of the general paralytic who died with a temperature of 109° F., certainly showed changes similar to those met with in the cortical cells of the other cases. This may have been due to the pyrexia, for it is unusual in general paralysis to find very marked changes in the chromatic substance of the anterior horn cells, and I have never before seen such universal affection. At the same time it must be remembered that in consequence of the venous hæmorrhage profound circulatory disturbances with venous congestion and stasis must have occurred in this case. (2) Exhaustion from discharge of energy in the production of the fits. If this were so, the anterior horn cells should be affected as much as the cortical cell; moreover, in the animals that died after ligature of vessels, the same changes were observed, whether they had fits prior to death or not. Again, in Case 4, I was unable to discover any appreciable difference in the appearance of the cells of the two hemispheres, although there were 400 left-sided fits. These facts point to local circulatory conditions being the main cause of the difference in the appearance presented by the cells of the cerebral cortex. Leonard Hill has shown that any increase in the venous blood in the closed cranial cavity is accompanied by a corresponding diminution of arterial blood, so that venous congestion, stasis and exudation, will cause arterio-capillary anæmia in the brain. But we have seen that the cells of the medulla do not suffer to anything like the same extent as the cells of the cortex. Consideration of the veins of the brain shows that the return of blood of nearly the whole external and the upper part of the internal surface of the hemisphere is by veins which run upwards to enter the longitudinal sinus. Backward pressure of venous blood would much more readily lead to stasis and congestion in these veins than elsewhere. Exudation would be more abundant, and carbonic acid tension in the regions which are drained by these veins, greater than elsewhere. Probably this increased CO_2 tension increases the excitability of the neurons.

It is suggested that the marked bio-chemical changes observed in the cortical pyramidal cells in the cases referred to, is due primarily to the alteration of the lymph environment. If anoxæmia does not of itself produce this change it is possible that increase of carbonic acid or other products in the lymph may be the principal cause; although it is not denied that insufficiency of oxygen and excessive activity may play important parts.

Changes in other tissues—such as striped muscle, heart muscle, liver and kidney. I have examined microscopically (staining by the Marchi method) these tissues in five fatal cases of status epilepticus, and in a large number of general paralytics who have died after prolonged epileptiform seizures, and have found that they are the seat of extensive early fatty degeneration; although to the naked eye, *post-mortem*, this condition would not usually be suspected. The cause of such marked fatty change in the muscles in cases of status epilepticus in persons who were a few days previously in good bodily health is difficult to explain. The fat must come from actual tissue waste with incomplete oxidation of the fat owing to venous condition of the blood. It is possible that fatigue products may play a part, for Araki, working in Hoppe-Seyler's laboratory has shown that lactic acid appears in the urine after prolonged epileptic convulsions.

Dr. F. E. BATTEN.—"Congenital abnormalities in the nervous systems of three children." (1) Non-development of left cerebral hemisphere (growth arrested about the 7th month of fœtal life), absence of left pyramid, absence of left fillet, diminution in size of right superior peduncle of cerebellum, left olive possibly a little smaller than right, cerebellum of normal size.

Inferior peduncles of cerebellum are of equal size, and posterior longitudinal bundles are normally developed. Below the decussation of the pyramids the cord appears almost normal, except that the right side is slightly smaller than the left.

The specimen was taken from a child aged 6 months, who had had fits since birth, and had congenital absence of the left thumb. The child had an obviously asymmetrical head, knee-jerks very active, equal; right arm-jerk greater than left. During a convulsion the eyes were turned to the right, the left leg drawn up, the left arm flexed, and the right leg rigidly extended.

(2) *Hydromyelia and congenital defect of the cerebellum* in a case of encephalocele of the occipital region from a child aged 4 weeks. The encephalocele was removed, and after death it was found that the cyst was in communication with the 4th ventricle.

The cerebellum was very defective, only the lower portion of the lateral lobes being normally developed. The middle lobe was absent. There was separation of the posterior columns extending through the cervical region, and from the 3rd to 9th dorsal segments, there was dilatation of the central canal, which was largest at the level of the 5th dorsal segment. The lumbar cord was normal.

(3) *Heterotopia* in the dorsal and cervical regions of the spinal cord, in association with spina bifida in a child aged $1\frac{4}{12}$.

Drs. H. H. Tooth and Risien Russell.—"Acute Myelitis with Bacteriological Examination." A man, aged 36 years, previously healthy with the exception of an attack of influenza four or five years before the illness which necessitated his admission into the National Hospital for the Paralysed and Epileptic, Queen Square. No direct or indirect history of syphilis or other venereal disease could be obtained. A month before death flaccid paraplegia of rapid onset, in which right foot at first escaped, ushered in by pain and tingling in the back, between the shoulders, radiating to front of abdomen and down inferior extremities, where there was also a subjective feeling of numbness. Temporary tingling and numbness in hands. Three weeks before this he felt out of sorts and languid, and experienced pain between shoulders radiating to front of abdomen. Movements of right arm and hand weaker than left, except at shoulder (patient right-handed). Diaphragm and intercostals normal. Left pupil larger than right, but they reacted normally. Slight comparative anæsthesia to light touches and pricks below level of third rib in front, and spine of scapula behind with inability to distinguish between heat and cold over blunted area, everything being called hot. Knee-jerks at first absent; subsequently elicited on reinforcement, though exceedingly feeble. Retention of urine, with overflow dribbling. Constipation. No bedsores, but blister on sole of right foot, result of a burn. Skin of dorsum of left toes glossy. Diminished reaction to faradism in muscles of lower extremities, and to galvanism, K.C.C. = A.C.C. in all muscles below knees except anterior tibial group on left side in which A.C.C. > K.C.C. Thigh muscles K.C.C. = A.C.C. and in some K.C.C. very slightly > A.C.C. Urine: a trace of albumin. Pyrexia—101° to 104° F. Developed severe diarrhœa, became collapsed and died.

At the autopsy cord of normal consistence throughout, but on section and subsequent microscopic examination marked evidences of disseminated myelitis with minute hæmorrhages detected. Three diplococci, closely similar morphologically, were cultivated from the substance of the spinal cord. Two of these resembled each other somewhat in their mode of growth on nutrient media, while the characters of the growth in the case of the third organism were entirely different from those of the other two. Inoculation experiments were conducted on the monkey, cat, guinea pig, and mouse, but only one of the diplococci proved pathogenic, and this only to the guinea pig.

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